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OF THE

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Preface.

THE Reports and Memoirs published in this volume represent a portion only of the actual "Transactions" of the Bristol Medico-Chirurgical Society. Some of the papers originally prepared for the Society, but subsequently published in different medical journals, have been omitted or presented in greatly abridged form. Moreover a large portion of the ordinary work of the Society, comprising discussions on given themes, or upon reported cases, or on morbid specimens exhibited at the several meetings of the Society during the years 1874-77 (inclusive) finds no other record than that contained in the minute book of the Society. In fact the opportunity of presenting a detailed report of all the transactions of the past four years has been lost by lapse of time, while from the same cause, the interest felt in proceedings which may now almost be termed posthumous, necessarily suffers a corresponding diminution. But notwithstanding that the material here collected appears also, in consequence of delayed publication, under relatively unfavourable circumstances, it is hoped that the small volume now put in circulation will be found to contain cases and observations of more than ephemeral interest, and worthy of a permanent place in our medical records. If the work be substantial it can scarcely be considered out of date within half the period assigned by the Horatian maxim "nono prematur in anno."

The issue of "Transactions," or any formal reports of proceedings, was not originally contemplated by the Society, since its work was at the commencement confessedly tentative, and its action concentrated on the purely practical object of furthering the local scientific status, and consolidating local professional interest, by the general and hearty co-operation of its members assembled at the regular evening gatherings. The success which attended their efforts led, however, in due course to a general desire that the work accomplished from session to session should be preserved in a more permanent and accessible form than the Secretary's minutes. And it was finally determined that a small volume containing such papers and reports from past years as would fairly represent the work accomplished should be published. The present change of plan has thus grown out of the past action of the Society as a natural sequence of its continuous progress and rising interest. The circumstance that some of the essays read before the Bristol Society have already found acceptance in Journals of long established reputation, seems further to justify the course now adopted. Other special grounds and inducements to the present undertaking, by which the Society though adding to its labours and responsibilities aims at their more complete fulfilment, are indicated in the following extract from an address delivered in the present year by one of its Presidents :—

"In suggesting a re-consideration of the proposal to publish our proceedings, it may be urged that we are now, after some years' experience, in a better position than at the commencement of our work to estimate the prospect before us. We possess a sufficient accumulation of material to start with; and we have a good assurance of a continuous supply of papers, discussions, and morbid specimens. We may also assume that local interests which have slumbered will again spring up in connection with a local volume of Medico-Chirurgical transactions. The members who have joined our Society at later periods might, presumably, wish to know more of the work done by our older associates.

Friends in the neighbourhood, or at a distance, who have formerly been connected with the medical establishments of the city, might receive with interest an occasional volume of essays, not intended to compete with weekly, monthly, or quarterly journals, nor to be filled with extracts of work elsewhere transacted and published, but simply to represent our centre of professional opinion and action. Such a volume might even be welcomed as a proof that the medical centre of the West of England maintains its claim to be considered a teaching power, fairly abreast with the general progress of science, and able to sustain its old repute."

In preparing for publication, however, it soon appeared that much excellent material which had been utilised in the discussions of the time could no longer be obtained. In particular it was no longer possible to make drawings of rare and interesting morbid specimens which had been exhibited; nor in many cases could sufficiently complete and exactly descriptive details of the *post mortem* appearances be collected. Reports have therefore been limited to such cases as were accompanied at the time by an accurate account of the morbid specimens, or of their clinical history, or by commentary on the pathological processes of which the specimen supplied typical examples, or lastly by a statement of the results of operative procedure. Any notable deficiencies in these respects are in great measure due to the circumstance that the Society did not undertake the publication of papers contributed, in consequence of which many communications were made by word of mouth, or from notes, the elaboration of which in a form suitable for publication, was hindered by press of later engagements. The Society has further to regret the loss by absence and decease of members who had taken an active part in its past proceedings; a loss which has become painfully evident in the attempt to gather together and preserve a record of experiences fully communicated at the time they were brought before the Society, but of which no complete account can be now recovered.

It seemed well, thus far, to allude to the circumstances under which the Society's first volume of "Transactions" came into

existence. But our retrospect (brief as it is) while serving to explain omissions and deficiencies which cannot at this late date be wholly rectified, naturally tends to quicken the critical sense with which the labourer reviews his own work. And it may be frankly admitted that, every allowance being made, there still remains ample room for improvement. Nevertheless the retrospect which leads to *self-criticism* is not devoid of happy results, when criticism means mental life and motion, as opposed to the petrifying influence of an uneventful routine. And whatever be the reader's estimate of the scientific or practical worth of the Society's work *outside* the immediate circle of its operation; there is *one* measure of its value on which we may be permitted to dwell for a moment, viz., the promise which it affords of future progress, under the educating power of a criticism exercised *within* that sphere. Our volume simply represents the current and, so to speak, incidental studies of members of a Society still passing through its early phases of development. It is not possible that a miscellaneous record which represents the work of many independent observers following out separately their particular opportunities, should exhibit in equal degree that logical method and singleness of aim which should distinguish an elaborate monograph containing the matured results of its author's persistent and undivided attention. Yet, however unpretending as a literary effort, the publication of Transactions by a Society marks its growing self-consciousness of present active power, and its faith in the present and prospective usefulness of such an undertaking. The self-criticism which it excites is also in itself a source of strength, and a powerful motive to further exertion, and may be reckoned a gain to any Society. Hence the truest justification of a Society's action in printing its "Transactions" lies rather in the internal evidence of usefulness than in any display of its labours before the public.

The mere act of publication affords indeed no proof of the powers and performances of a Society. Nor can its success before

the public be tested otherwise than by outside criticism, and the ratification of public judgment. The appeal implied in printing Transactions, indicates therefore, the intention and wish of a Society to be so judged. So far at least as the present volume can pretend to challenge criticism from without, we cordially accept it, and entertain, beside the hope of an encouraging verdict, the firm conviction that we cannot but gain help and instruction from the corrective action of an enlightened criticism from without, which must needs carry with it a higher issue than does the narrower and rarely impartial exercise of self-judgment. For he who looks only into the depths of his own experience often sees nothing but his own reflection on the surface; while the inner truth which he seeks is attainable only by a deeper and independent insight into the general current of thought, and by tracing it back through the troubled waters to its primary sources beyond the time and circumstance of any single observer's action.

But here we reach that delicate ground of "original claim," towards which author, critic, and general public alike, gravitate, where the former finds himself confronting both critic and general reader, and is tempted to employ the customary "preface" as a recognised vantage ground of defence, forearming himself against anticipated attack. We shall venture to assume that we do not overstep the limits of prefatory discourse in offering a few observations on the science of criticism as applicable to medicine.

Happily, the critic of medical science is not represented in the definition, given by a renowned statesman, of the art-critic*, and yet his function may be exercised with widely different effect and value acccording as he, in passing judgment upon the work of any particular writer, discusses merely his style, method, and opinions, or endeavours to assign to the subject which he has handled, that position of advance which he has won (or failed to

* "Who are the critics? The men who have failed in literature and art."—"*Lothair*," *by Disraeli.*

win) for it, in the general aggregate of scientific truth. For it is not enough for the scientific truth that each person, or even age, shall criticise his, or its, own work alone. The higher function of criticism is to grasp the whole situation rather than to limit itself to particular facts and beliefs, or the mode of investigation and reasoning of any single individual or generation. It cannot ignore the conclusions it has inherited, though always ready to reconsider them, and reject what is untrustworthy. Neither can it consent to be deprived of the clearer perception which breaks in upon the mind when new facts are tested by old experiences, or when old experiences are tested by new facts. In a word, an enduring criticism does not lend itself to the momentary needs of a transition period; nor in seeking the truth of a fact or argument, does it question its age or its youth, but endeavours to recall its whole history, and if that history be traceable to remote times, those times form as necessary an element of the investigation as the most recent event connected with it. Practically, therefore, the latest medical doctrine is a perpetually renewed verdict on ancient doctrine, with additions, which in their turn, are put on their trial before a critical sense whose cultivation keeps pace with increasing experience: for *cæteris paribus* the accuracy of our judgment may be said to bear a definite relation to length of experience.

Hence the best work of the investigator and critic, is truly correlative;* each at times exercising the function of the other, while their respective contributions form integral parts of the whole aggregate of scientific truth at which each is aiming. For the exercise of critical faculties is no exclusive possession of the professional critic, but an equal and common right of all engaged in the same studies. And further, there could be no real progress without the common and salutary operation of a criticism which is

* It seems scarcely necessary to point out that no allusion to the present fashion of "reviewing" is here intended.

positive and constructive, as well as that which is negative and simply aims to purify by elimination. Considered from this point of view, the critical judgment of his peers must and always will be acceptable to every earnest enquirer, though he may reasonably demur to the bare assertion of doctrinal, or individual authority when arrayed adversely against facts and experiences which he has endeavoured to establish by painstaking and careful work. The appeal to personal authority in confirmation, or negation of facts or opinions, is only admissable when special proof of competence has been already given, and only then so long as the verdict pronounced is reconcilable with the experience we have gained for ourselves. And since the value of authority is entirely relative, it follows that the best mode of raising this value, and of guarding against the misuse of authority (whether of general doctrine or individual experience) is to multiply our own self-experience. In so doing, our own senses and powers of thought become a law to ourselves, a rightful authority for our beliefs ("*mens sibi conscia*!")

Nevertheless this critical sense, the exercise of which is not only a right, but also a duty of all students of science, is more truly an outgrowth of their aggregate labours, than the fruit of any single mind. It has been justly said that the medical man reflects the scientific opinion of his time. If, by individual stretch of thought, one or another gain a step in advance of his neighbour, his success is mainly due to the use which he makes of facts and premises which are the common property of his generation. If again, by his labour he enrich the whole community, he but repays the debt of intellectual influences by which he is surrounded, and of sympathies by which he has been borne onwards so long as his mind kept contact with the aspirations of his compeers. And as with the individual so with the community. The latest generation—inheritors of that great Past which is to the Present as ages are to days—is indebted for its accumulated knowledge (and capacity to acquire knowledge) to the labour and struggle of

preceding generations: is indebted also for its present cultivated critical sense (which it is too apt to assume as its own superior endowment!) to that universal mental energy which has bred a perpetual desire to explain and interpret.

And again, as with the growth of any particular knowledge, so it is with knowledge in general. Each department of science has its reciprocal relations with the other, lending and receiving aid from all sides; yet so, that each preserves its own individual form and progress. Thus medical science while conforming to the standard of general culture by which it is surrounded, pursues separately its own methods and ends: not indeed without frequent risk of being diverted from its true course by the glare of discoveries and revelations in collateral branches of science. But when knowledge is being rapidly accumulated and opposing views obtain various degrees of assent, the conflict of opinions necessitate incessant re-examination of the grounds upon which they are formed. Meanwhile so long as the demands of medical art compel the physician to make use of any generalisation which the science of his day permits, he must needs persist in his search for principles (or at least postulates) which he can turn to account. Thus arises system after system according as medical opinion falls under the sway of physical, physiological or pathological doctrines, to be rejected as soon as any fresh discovery (or what passes for such!) seems to disprove the last, or open out a new prospect for future belief. With the rise and fall of theories thus hastily propounded, it cannot but happen that the dogma of to-day proves to be an exploded fallacy to-morrow. But it survives, nevertheless, in the minds of those who have other occupation than that of steadfastly dogging the heels of scientific progress. Perhaps at no epoch has there been so complete a sweep of old opinion, and so universal acceptance of new, as to leave no leaven of the past in the innovation of the present. While at every epoch the revival of old modes of thought masked only by a setting of modern phrases and terms, may be traced to a striking extent in the newest

hypothesis. And so again, that which is really new often fails to be clearly grasped because it has been confounded with some tradition which has not been entirely swept away into the medical dust-hole, but has gradually changed its face as its original basis of fact and belief has been replaced by more accurate observation.

Thus the history of medicine reveals to us a series of recantations of doctrines held for a while, then changed for others, which in their turn give way to still later novelties. And as it would seem the "principles" on which these various medical systems have rested are more often the fancies of prepossessed minds than deductions from actual experience. Need, therefore, and ample opportunity for that science of criticism whose aim is to re-compose the discordant creeds and embarrassing claims of so many systems, authorities, and schools.

But when, in the exercise of our critical expositions, we deal with the facts and evidences of any scientific position which we or others may seek to establish, that form of criticism must surely be most suitable which merges the personal opinion of the critic in some larger rule of experience, and tests the author's conclusions by applying still wider generalisations rather than questioning his competency. Judge-made law, rarely in any case acceptable, is still less so when pronounced by the critic of medical science; for unfortunately medicine with its numberless systems has yet no universally recognised code, and with its numberless authorities engaged in upholding or subverting these systems, has yet no infallible judge; nor amongst numberless critics can there be any whose judgments reach beyond present knowledge, or many whose judgments fully come up to the present standard in all points. And so long as the practice, precedents, and principles of medical science are alike mutable and variable, the most becoming characteristic of contemporary science should be—its moderation! Since in the conflict of system and pre-existent authority with individual freedom of observation the latter must necessarily be overweighted, and too often sacrificed to arbitrary laws which may themselves be

little more than guesses at truth, though putting on for a moment the semblance of final deductions of science.

We conclude our prefatory remarks with a homeward glance, and venture to offer another extract from the address to which reference has been already made.—

"Looking at the work done, we cannot but admit that greater variety of contribution to practical medicine and therapeutics, as well as the general principles of pathology, would be acceptable. It is indeed an invaluable privilege for us to see and hear all that can be reported from our clinical wards, our operating theatres, and our post-mortem rooms. For such experience is the foundation of our knowledge. But I am tempted to ask—Is this knowledge to end in the mere accumulation of facts? Are we too busy in our search for facts to allow the older traditions to challenge our attention? If we reject the generalisation of yesterday we begin another to-day, for the human mind never ceases its efforts to explain; and so the latest fact becomes the basis of the last theory, until both narrow down to the meanest dimensions. Now I submit that under such circumstances a little conservatism is excusable. Should it not be our care to set ourselves sometimes to the task of thinking over those higher medical reasonings on which our medical insight depends? It cannot be that we intentionally exclude from the sphere of our work the study of those generalisations on the faith of which the practitioner draws conclusions and practical rules for himself in his daily observation of disease. The problems which he is called upon to solve upon the spur of the moment are too often under-estimated as merely therapeutical. But therapeutical science demands, at one time, a complex application of physiological principles; at another time, the closest attention to the most profound deductions of pathology (for diagnosis and treatment are now inseparable); at a third moment, resting upon its own special experience, its discoveries reflect light upon either or both of the former methods; while at all times it remains a high accomplishment of our medical art.

If morbid anatomy can prosper better by giving up theory, medicine *cannot*, nor can a medico-chirurgical society."

And, lastly, if it be permitted to include within the range of our proem observations and hints of such various import, we would address a word in season to our colleagues of the Bristol Medico-Chirurgical Society.

Every society that pursues purely scientific aims, and depends solely upon voluntary effort, labours of necessity under the want of fixed constitution, and has an uncertain vitality. It is thus in constant need of sustained and extended support from the whole body, as well as from its more prominent members. And as it is important to keep this consideration always in mind, your Council feel it an imperative duty to forewarn the society against that inaction which is so perilous to its stability. While, therefore, we look back with satisfaction on the successes achieved in past years, there is always reasonable ground for urging upon all and each of our members the duty of active co-operation. In the time of prosperity they feel it but prudent neither to slacken in the discharge of their own responsibilities, nor to omit such urgent persuasion as may be allowed in dwelling on the work and prospects of the future. The time has not arrived, and ought not to arrive, when our Society may "rest and be thankful." Let earnest laborers extend in unbroken succession the field of their investigations, that the harvest may abound more and more. Let their aims rise higher as they watch the ripening fruit in fields already successfully tilled. Let each individual effort stimulate to general exertion. Let us adopt the noble thought of a truly noble poet, who in speaking to his countrymen well understands their mind and temper:

> Men my brothers, men the workers,
> Ever working something new;
> What they have done but the earnest
> Of the things which they shall do.

September, 1878. H. E. FRIPP.

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ORIGINAL MEMOIRS.

Abstract of paper on the Physiology of general Paralysis of the Insane, and on Epilepsy.

GEORGE THOMPSON.

THE author of this paper brought forward certain theories, and afterwards the facts supporting them :—

The Theories.—(1.) That the organic change which exists in the very early stages of General Paralysis consists of a diminished calibre of the vessels, which is of the nature of a persistent spasm, (2.) That this spasm, though persistent if left untreated, is, if recognised early, amenable to remedial means. (3.) That the lesions found after death are not the cause, but the result, of early organic changes that need be only of temporary duration.

The Facts.—(1.) That the tracing of the pulse taken at the wrist by the sphygmograph in cases of General Paralysis, when untreated, is precisely similar to that found in a person in good health, who had been exposed to a cold bath for the space of one minute. (2.) That the vessels of the retinæ and optic discs are thin and attenuated, and the discs themselves are void of their natural pink tint. (3.) That General Paralytics are more frequently the subjects of cerebral syncope than persons labouring under any other disease of the brain. (4.) That in the early stages of General Paralysis the temperature of the body is lower than in health, and the skin of persons so affected is then in that condition

N.B.—This paper, as given before the Society, was published in the January and April numbers, 1875, of the "Journal of Mental Science," to which Journal we refer our readers for further details.

known as the *cutis anserina*, resembling that condition seen in the cold stage of ague, in cholera, or in the rigors preceding a febrile attack. (5.) That by the administration of such remedies as are known to be antagonistic to spasmodic action, the pulse-tracing may be brought back to a healthy form, the natural appearance of the retinæ may be regained, the temperature of the body may be raised to the normal standard, and then the skin will assume its original smoothness.

The accompanying photo. sphygmograms illustrate that portion of the paper which refers to General Paralysis of the insane.

No. 1. Henry Batt is a typical illustration of the "pulsus tendus" seen in the earliest recognised stages of the disease.

No 2. William Jennings, "12 noon," is another illustration of the same disease at a later stage.

No. 3. William Jennings, 3 p.m., shews the high susceptibility of the system in G. P. to the influence of a meal containing half-a-pint of moderately strong ale,—general hypersensibility of the nervous system to stimulants.

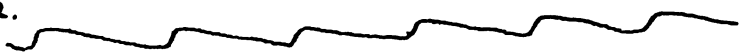
The author explains the occurrence of epileptic seizures in this way. There is a sclerosed neuroglia, whether the result of the insane diathesis, whatever that may be, or the result of a specific disease, such as, say, syphilis. This hardened neuroglia is no longer able to accommodate itself to the frequent, and often sudden, changes in the quantity of blood passing through it, and the least resisting portion of the brain substance—the cells and nerves—is suddenly exposed to an unusual degree of pressure. Now, as the various centres are subject to varying degrees of hardening, as of varying degrees of blood-flow into and through them, so are the phenomena which follow upon the disturbance varying in character. But I would remark here that there is seldom any degree of change of phenomena in individual cases. This will explain how it is that fits as they occur in the same individual, possess such a remarkable resemblance the one to the other; the cry is the same; the manner of falling is the same—so that often the part once

Sphygmo-graphic tracings, copied from photo-graphs, illustrating Mr. Thompson's paper on General Paralysis.

No. 1.

Henry Batt. Dec. 1st 74. 120 grs.
Temp. 98°.4. Pulse 54

No. 2.



Wm Jennings. Oct. 22/75. 12 noon
Temp. 96.6. Pulse 64 150 grs.

No. 3.

Wm Jennings. Oct 22nd 75. 3 p.m.

bruised in a bad epileptic never heals, and is always receiving further laceration; the character and degree of the muscular spasm hardly ever varies—if once unilateral, always unilateral; the period of unconsciousness (if this occurs) is always of the same length; and the mental disturbance will vary as little as the other phenomena.

The treatment of Epilepsy is summed up as follows:—

It is a singular fact that all medicines that possess the confidence of the profession as being antagonistic to convulsive action are such as are known to control the vascular system. Ergot of rye, belladonna, bromides of potassium and ammonium, valerian, and its combinations with zinc, are familiar instances. I was once so fortunate as to commence a sphygmographic tracing, which began with a form modified by ergot, and ended with the characteristic tracing found in epilepsy; the patient in the ten seconds required to pass the slide over the clock-work box of the instrument having gone off into a severe fit. I would not rest contented, however, in knowing that certain drugs have a kind of "governor-ball" action over the vessels, for at the best treatment by such drugs can only be deemed as palliative. Such treatment may ward off the fits until the "epileptic habit" has become accustomed to that form of treatment, when, as we often see, the fits return with an accumulated force. The treatment, which is to be of a lasting and effectual character, is that which shall have for its basis a means of attacking some constitutional habit or condition. If that habit be, say, syphilitic, then it is reasonable to suppose that iodide of potassium will do more than "ward off" the fits; if strumous, then cod-liver oil will have a marked effect; if "herpetic," arsenic should be given; and so on.

December, 1874.

Observations on a case of Ossification and Necrosis of the Laryngeal Cartilages.—Operation of Tracheotomy, and discharge of the sequestrum through the mouth.

W. J. Fyffe, M.D.

JAMES CURTIS, a private of the 73rd Regiment, aged 37, was admitted into the Royal Victoria Hospital, Netley, on the 12th December, 1871. He had completed nearly twenty years' service, of which he had served five years at the Cape of Good Hope, and three years in India, including a period of several months during which he was engaged on active service in the field in the suppression of the Sepoy Mutiny, in 1857-8.

While serving in Portsmouth, in 1866, he contracted a venereal sore. The character of this sore was not stated in his official medical documents, and it is therefore impossible to say if it was a true syphilitic chancre or a soft non-infecting sore; but it does not appear to have been followed by secondary symptoms of any kind.

Four years subsequently, namely, in June 1870, while in India, he began to suffer from hoarseness, loss of voice, and *dysphagia*. These symptoms were soon after followed by the formation of a large abscess in front of the neck. The abscess was described as having been as large as the closed fist, and as occupying the space between the lower jaw and the middle of the thyroid cartilage, and bulging as far forward as the chin. It was extremely tender and painful. This abscess was opened in two places: one incision was made to the right of the thyroid cartilage, on a level with the pomum Adami; the other opening was made at the inner edge of the sterno-mastoid muscle, about an inch above the clavicle. A large quantity of pus was discharged, but the opening of the abscess was not attended with any alleviation of the symptoms of aphonia and dysphagia. All traces of the abscess disappeared, and the incisions healed up. In this state he was sent home to England.

His condition on arrival at Netley was as follows:—There was great difficulty of breathing, evidently produced by mechanical obstruction in the larynx. Each effort to inspire was attended with a harsh crowing noise. The voice was reduced to a whisper. He was unable to lie down. The countenance was anxious, but there was no abnormal rapidity of pulse or increase of temperature. In other respects he was a healthy looking man, with florid complexion, a well nourished body, and no sign, either physical or general, of lung disease. On examination of the external parts of the neck there appeared to be some thickening of the skin over the thyroid and cricoid cartilages, probably the result of the abscess already alluded to. Some irregularities on the surface of the right ala of the thyroid could be felt,—one distinct depression, indicating a loss of substance, was detected, corresponding to the position of the incision made for the discharge of the abscess. No pain or tenderness could be observed over any part of the cartilaginous skeleton of the larynx.

I should observe there was a constant short stridulous cough, without expectoration.

On examination with the laryngoscope the aryteno-epiglottidean folds were found red and swollen; the rima was irregular in shape; the right arytenoid cartilage seemed to be pushed over towards the left side; the rima, however, opened fairly on inspiration. There was want of approximation of the true cords. While they separated during inspiration a whitish tumor could be seen between them, apparently protruding from below. As the patient was in much distress it was difficult to obtain a good view of the larynx.

The case being urgent, everything was prepared for the operation of tracheotomy in the event of any paroxysm of dyspnœa supervening. The symptoms having become very alarming in the middle of the night, the operation of tracheotomy was performed by Surgeon Major Mackinnon, C.B.

The operation was followed by instant relief to the patient. He

breathed quietly and easily through the tube. Several small pieces of lymph were discharged through the canula after its introduction.

For a week he progressed very favourably. Seven days after the operation he coughed up suddenly a piece of bone, which was very fœtid, and presented the usual character of sequestrum. This appeared to relieve him still more. Some purulent matter passed away by the mouth and through the canula, but this soon ceased. For two months after the operation he was in a very satisfactory state. His general health improved, and there was no sign whatever of lung complication. The canula was removed from the trachea, and the wound was allowed to heal. At this time laryngoscopic examination revealed some thickening of the superior false vocal cords and want of approximation of the true cords. The voice was not restored: it was frequently whispering, sometimes stridulous and metallic. No symptoms or signs of lung disease could be discovered. The man appeared to be on the fair way to recovery, and was in excellent bodily health and high spirits.

He was, however, suddenly attacked again with urgent laryngeal obstruction, and it was found necessary to re-open the trachea, which proceeding, as on the first occasion, at once relieved him. Whether this second onset of symptoms rendering tracheotomy a second time necessary was the result of further commencing necrosis, of stricture, or of spasm acting upon a rima already to some extent narrowed, I found it difficult to say, as it was very difficult in this patient to see the condition of the parts about and below the vocal cords. He continued to wear the canula; and in the course of a few weeks was discharged from the Hospital and from Her Majesty's service.

He was admitted at my request by Dr. Morel Mackenzie into the Hospital for Diseases of the Throat, in Golden Square. He did not undergo any particular treatment there, and subsequently took up his residence in Winchester, where he died very soon

after, suddenly. Unfortunately, there was no post mortem examination; the case, therefore, is incomplete in this respect.

The points of interest which have occurred to me in this case are these: Do ossification and necrosis of portions of the laryngeal cartilages occur as an independent disease—independent, I mean, of what is generally called laryngeal phthisis, or of pulmonary phthisis? In this case I was unable to discover any evidence of lung disease whatever.

As there appears to be some difference of opinion among pathologists in reference to the connection between laryngeal and pulmonary disease, I will quote a few authorities on the subject.

Dr. Fuller, for example, considers there is no such disease as laryngeal phthisis independent of tubercular disease of the lungs.

*Mr. Porter describes two diseases to which the cartilages are liable. One he calls mortification of cartilage, where portions are found dead and blackened but not ossified. This is usually accompanied by abscess, which points either externally in the neck or bursts into the larynx, trachea, or œsophagus. This is a very rare disease. The other affection he describes as true phthisis laryngea, arising from degeneration of one or more of the cartilages or parts of them into bone.

This osseous change takes place according to the author about the age of 32. It is an extremely insidious disease, very slow in progress, and causes at first little or no inconvenience to the patient, hoarseness and dysphagia being generally the first symptoms. As the degeneration increases inflammation sets in, followed by abscess. The ossified parts become necrosed. Ulceration, thickening and corrugation of the mucous membrane result. The abscess may discharge externally, or by ulceration into the larynx, and portions of the necrosed cartilages are coughed up and discharged, as in this case, by the mouth. Mr. Porter did not regard phthisis laryngea, as he thus describes it, as resulting from

* "Diseases of the Larynx, 1837."

the previous existence of disease within the chest, and states that it would be difficult in any case to place the two affections in the relation of cause and effect.

Rokitansky regards ossification of the cartilages of the larynx as a disease principally of advanced life. He describes a form of suppurative inflammation of the larynx resulting from peri-chondritis. The peri-chondrium becomes detached, either in isolated spots or over the entire surface of one of the cartilages, and here the cartilage is found villous, gritty, and necrosed. This may result from a rheumatic origin, or as a consequence of the exanthemata, or from syphilis, or the abuse of mercury.

Dr. Morel Mackenzie regards ossification and necrosis of the cartilages more as the result than the cause of laryngeal phthisis, and after defining that disease as chronic thickening and ulceration of the larynx (usually occurring consecutively to pulmonary phthisis, but sometimes being present before lung disease is evidenced) he points out that ossification and necrosis of the cartilages more often result from phthisis laryngea than from all other diseases. With regard to the relation existing between laryngeal phthisis and pulmonary phthisis, Dr. Mackenzie has only met with three cases in which, on post mortem examination, the former existed without the latter. Mr. Porter, however, gives the details of several cases of extensive laryngeal disease in which there was no sign of lung disease whatever.

Niemeyer, in his chapter on Tubercular Ulceration of the Larynx, observes that this form of laryngeal disease, although rarely occurring as an independent malady, is one of the commonest complications of pulmonary consumption, and he seems to regard it as the result of innoculation. The mucous membrane of the larynx becoming strained and abraded by constant coughing, affords an exposed surface, which readily absorbs any caseous material voided in the sputum. Ossification and necrosis, he observes, are frequently combined with tuberculosis of the larynx; but he also admits that these pathological conditions may result as

an independent malady from peri-chondritis. Although this may occur in robust and otherwise healthy subjects, yet such conditions are chiefly to be found in persons tainted with syphilis or by the abuse of mercury.

The inter-dependence between ulcerative or suppurative affections of the larynx and lesions of the lungs is a question of some importance. The probabilities of local pyæmia occurring in such cases has been pointed out recently by Dr. Geo. Johnson, in a lecture on the relation between laryngeal and pulmonary disease, founded on a case of pneumonia following syphilitic ulceration of the larynx; and he expresses his belief that the pneumonia was the result of transference of morbid material from the laryngeal ulceration to the minute bronchial tubes. Similar results may follow in the acute specific diseases, such as diphtheria, as the result of transplanting of morbid products from the throat to the lungs. If this be true as regards the acute diseases, it is also doubtless true in reference to the chronic forms. The vascular connection between the lungs and the larynx is so intimate, and the air communication between them so constant, that one set of organs cannot suffer long without involving the other. But there certainly do appear to be cases similar in character to this one where the laryngeal disease appears to have commenced as a purely local affection resulting in abscess, and finally terminating in partial ossification, necrosis, and exfoliation, and this without lung complication.

In reference to the operations for tracheotomy which were performed, I confess I think it would have been wiser to have left the canula in the trachea after the first operation, and not to have allowed the opening to heal. I think that in all probability the removal of the tube and healing of the wound led to the formation of a laryngeal stricture, which rendered a second operation necessary.

January, 1875.

Croton-Chloral in Neuralgia.

E. Markham Skerritt, M.D., M.R.C.P.

THIS paper was illustrated by the details of numerous cases treated at the Bristol General Hospital.

It had been shown by Dr. Oscar Liebreich, that croton-chloral was physiologically an anæsthetic, and in large doses a hypnotic; that anæsthesis always began at the head, and only after this region was fully under the influence of the drug and sleep had been induced, did the anæsthetic effect gradually extend over the rest of the body. With large doses the respiration and pulse became slower, and death was due to arrest of the respiratory movements: in this respect croton-chloral differed from chloral, as with the latter death occurred directly from cessation of the heart's action. From the above, it would be expected that the most marked therapeutic effects of croton-chloral would be seen in the case of painful affections of the head.

The author had used croton-chloral in cases which might be arranged under the following divisions:—

(A) *Pain or other abnormal sensations about the face, head, and neck.*

(a.) *Ordinary headache*—varying from slight attacks to very severe, the worst forms being known as megrim or migraine, and popularly as sick-headache, bilious headache, or bilious attack. The affection in all these cases was believed to be neuralgic, and the nerve usually at fault was the first or ophthalmic division of the fifth. Migraine was an important variety, and it could not be too distinctly remembered that it was a pure neurosis, and was not due to digestive disturbance; the act of vomiting was in fact only an expression of the intensity of the nervous

exhaustion; and this act might be associated with severe neuralgia of other parts of the body than the head. Dr. Anstie recorded cases of intercostal neuralgia and of neuralgia of the testis, in which the attacks sometimes culminated in vomiting.

Case.—Migraine. Female, aged 45. Subject to attacks of "the bile," *i.e.*, severe headache, going on to nausea, and sometimes to vomiting. Between these attacks of "bile" she often had less severe headaches. After taking croton-chloral for a week, she said she had had no attack of the bile, but had been troubled with the headache; that is, the neuralgia had lost its intensity. In another week the attacks of headache were very much less frequent and less severe, and in another week, she was quite free from all pain.

Case.—Female, aged 24. For six or seven months subject to attacks of pain in the temples, coming on for about half an hour at a time, and then leaving her giddy, with a peculiar taste in the mouth: this would occur several times a day. After taking the drug for a week the headache was less severe, less frequent, lasted a shorter time, and left no giddiness behind. In another fortnight the pain had entirely left.

Case.—Female, age 16. For three months had had severe pain in left temple, coming on very frequently during the day, with a sensation of great heat of head, and lasting for about ten minutes at a time. Cured after two or three doses.

Case—Of interest as showing the association of vomiting with neuralgia of traunatic origin, and the effect of the drug upon it. Female, aged 22. For some years subject to headache, sometimes very severe; it always began over the right eye, in the scar of a cut

received when twelve years old. For the last two years the pain had been very great; it was sometimes accompanied by severe vomiting. There was a crop of herpes over the right eye. The patient came back in a week, and said she had had scarcely any pain since she had taken the medicine. The association of neuralgia with herpes, and of herpes with an abnormal state of the adjacent nerve, was well known; hence in this case the neuralgia was due to the abnormal condition of the nerve consequent upon the wound, and the ultimate cause of the vomiting was this local lesion.

Case.—Male, age 33, For *three years* had suffered from pain in the head, beginning at the occiput, and running forwards on either side. In a week he was very much relieved, and soon the headache was entirely removed.

Case.—Male, age 40. For six months had been troubled with darting pain in the left temple, leaving the scalp extremely tender; he could not lie on either side to sleep, as his "ears got numbed." In a fortnight he was cured. In his gratitude he stated that he had been treated at another institution for three months without any relief (and had doubtless had all the ordinary remedies); that he had been unable to take his food or to read, whereas, now, his appetite was good, he could read small print without any trouble, and had not felt so well for twelve months.

Case.—A boy, aged 10. Enlarged glands in the neck, ecthyma about the head, altogether much out of health; complained of frontal headache. The phosphate of iron had no effect on the pain, but croton-chloral removed it entirely.

(b.) *Facial neuralgia and toothache.* The success of croton-

chloral in this class of cases was not so marked when the pain was dependent upon the presence of carious teeth, as in those where no such definite local lesion caused the neuralgia.

Case.—Female, age 35, Much troubled with face-ache, connected with very bad teeth. In a week she was very much relieved, but had some headache and giddiness. An additional dose, *per diem*, soon cured both the face-ache and the headache, and the patient said her head had not been so well for months.

(c.) *Headache from inter-cranial disease.* Cases where this was present were not relieved.

(d.) *Sensations such as giddiness in the head without actual pain.*

Case.—Female, age 28. Complained of heaviness in the head, no actual pain, followed by soreness at the vortex. Very much relieved in one week.

Case.—For the notes of this the author was indebted to his friend Mr. A. Pearce Gould, of London. Female, age 22. For six months had suffered from constant giddiness, lasting all day, not influenced by position, exercise, or food, but going away on lying down at night. No pain—no other complaint—no marked anæmia. Had been under treatment for six months, without benefit. Bromide of potassium, and this combined with iron, produced no improvement. Ordered croton-chloral. For the next fortnight there was no improvement; on some days the giddiness was quite absent—then it became so slight as to be unnoticed when the patient was occupied, and she said she felt "quite a different creature." Croton-chloral was then omitted, and the giddiness returned almost as bad as ever. A further short course of the drug cured her.

(B.) *Pain in other regions.* In sciatica, intercostal neuralgia, pleurodynia and lumbago, rheumatic affections, dysmenorrhœa, and pain connected with digestion, there had been no definite improvement from the use of the drug.

Dose and mode of administration. In all the above cases the dose was five grains three times a day, except where otherwise specified; and patients unrelieved by this dose were often much benefited by the addition of another dose of five grains *per diem.* In private practice the same dose might be given every two, three, or four hours, according to the urgency of the case. Large or frequent doses might be given without danger.

Croton-chloral was but sparingly soluble in water, but a solution of ten grains to the ounce could be made if sufficient time was allowed, and warm water would dissolve the same amount more quickly. Glycerine made it rather more soluble. It might also be given in the form of pills.

The only unpleasant effects observed had been the following:—In two cases, vomiting; in one this was so constant after the dose that the medicine had to be discontinued; in the other it ceased when it was taken immediately after meals, and the desired result was obtained. In several cases, drowsiness, but not to any marked degree. In two or three cases, giddiness. In one, headache.

From the above details it would be seen that in the author's experience, the effects of croton-chloral had been such as might be anticipated from a consideration of its physiological action;—as the primary effect was shown in producing anæsthesia of the head, and only after this was well marked did its influence extend to other regions, so its remedial value had seemed to be almost limited to painful affections of the head. This conclusion, though at variance with not a few reported results, was in accordance with the experience of Dr. Ringer.

In the author's hands croton-chloral had been of the greatest use in neuralgias of the fifth nerve, and had appeared in many cases to act as a specific. Its effect could not, however, always be relied on, and certain conditions appeared more favourable to its success

than others; thus, the most marked benefit had followed its use in the neuralgias of young or comparatively young patients, especially in the headaches of anæmic women and girls. In these cases there had been cure or relief in 86 per cent. of the cases treated. About the climacteric period the success had fallen to about 50 per cent., while in later life there had again been a rise to about 60 per cent. At the climacteric period bromide of potassium was more reliable in its action. Again, when the headache had occurred in patients with marked hysterical symptoms, the result had not been nearly so favourable.

In order to obtain any marked or permanent effect on the headache, it was in many cases necessary to continue the use of the drug for some weeks, although in others a few doses produced a marvellous effect.

It was, of course, necessary to remember that to give croton-chloral for the neuralgic headache of anæmia, was to treat only one symptom of the anæmic condition, and that attention must also be directed to the removal of that state. Although, however, there was no evidence that this remedy could be considered as a direct tonic, yet it was certain that, in many cases, patients improved in their general health while taking it; it was an *indirect* tonic, and acted by the removal of what perhaps, more than anything else conduced to anæmia and mal-nutrition—that was, frequent or constant *pain*. Doubtless, *when* the general health had been improved by tonics, the neuralgia would in many cases disappear. But the special advantage of croton-chloral was that it would often quickly remove that which chiefly distressed and wearied the patient; in not a few instances where tonics had improved the general condition, but had left the headache untouched, croton-chloral had quickly cured this, and a too speedy substitution of tonics for croton-chloral had been followed by a return of the pain. The two kinds of remedies might advantageously be given at the same time, so that the croton-chloral might keep the neuralgia in check, while the tonic attacked the original cause of the trouble.

March, 1876.

The Chemical and Therapeutic relations of Salicin and Salicylic Acid as remedies for Acute Rheumatism.

W. H. Spencer, M.A., M.D. Cantab.

IT is little more than three years since the medical world was startled by the advent, almost at the same time, of salicin and salicylic acid as remedies for the cure of acute rheumatism. Both remedies were reputed, on no mean authority, to exert in acute rheumatism an influence for good such as no other remedies had been known to exert. The effects claimed for each were little less than specific. The advent of two different remedies with such high pretensions could not fail to awaken an eager interest in their use, and to divide opinion as to their relative merits. Almost from the first a preference for salicylic acid over salicin was clearly expressed,—at least, by those who publish opinions. Salicin was not to be compared with salicylic acid for its remedial effects in rheumatism. This was and still is the verdict of a wide clinical experience,—an experience, it is true, of no long standing. But salicin has always had some advocates, and it has them now; and Dr. Senator of Berlin has exceeded all others in his advocacy of salicin: he has derived an explanation of the good effects he and others claim for this remedy from certain chemical relations between it and salicylic acid. He contends that the action of salicin must be fundamentally the same as that of salicylic acid, for that salicin in the body is nothing less than salicylic acid itself. The attestation of a wide clinical experience in favour of salicylic acid, and the counter advocacy of salicin, may be, in Senator's view, both allowed; for both may unite and find a common explanation in the theory that salicin becomes converted into salicylic acid in the blood, and the active therapeutic agent being thus the same for both remedies, the effects produced by these should be similar, and salicin should have a common utility with salicylic acid in the treatment of acute rheumatism.

The question, therefore, from this new stand-point is no longer whether salicin may be better or worse than salicylic acid, nor, immediately, for what cases the one may be better adapted than the other: the question as between these two becomes a discussion and criticism of Senator's theory of common utility in chemical identity. I propose, then, to discuss the chemical and therapeutic relations of salicin and salicylic acid from the new stand-point, and this in a criticism of Senator's theory; for it seems to me that the theory itself is such in its circumstances and in its basis as to demand a full criticism in the interests of scientific medicine.

Medicine is largely dependent on other sciences for aid in working out its various problems. It works largely by means of theory, and much by theory founded on inferences drawn from principles which have been established within the area of other sciences, and by those who work within those other limits. The generalisations arrived at by other sciences—each of its own facts, and by the common methods—may be properly borrowed by medicine and made use of in its own operations. But it is just here that medicine needs to exercise a jealous care lest in the transfer and subsequent use of what is borrowed its scientific character be impaired. Medicine may be a peculiar and special department of knowledge: it may have a special field in which to work and work of a special kind to do within its area, and it may be by this marked off from all other departments of knowledge. Be the material and limits of any science what they may—these give not the title to be called a science, but the scientific character of the work. Medicine is always a science, and in company with the rest, when it works in the same paths and by the same methods as are deemed the only safe paths and sure methods for every other department of knowledge, and it is never scientific when it forgets to work thus. And it is in a particular department of medicine—therapeutics, to wit—that a jealous regard for the scientific character is needed most; for of all the yearnings of the

medical mind, the desire to exchange blind empiricism for a rational treatment of disease is perhaps the greatest. And in satisfying this desire, the inducement and the facility are singularly active to allure us into short and bye-paths, and to smooth for us the rigours of a strict scientific method.

In the present state of the sciences chemistry and physiology, we must meet in these with many transition-theories and much half-knowledge,—good, no doubt, so far as they go and whilst still held in place within these sciences; but these are not for medicine to borrow or to use. Wherefore, any theory brought up into therapeutics, and appealing to these sciences for a basis, must not refuse the closest criticism, lest perchance it should impair, in itself or in its consequences, the scientific character of medicine. Nor must it be forgotten that he who brings up a theory into therapeutics does more than make a suggestion to cautious minds able, by opportunity, to verify its facts and work out its consequences for themselves.

On such grounds, then, I propose in this paper to criticise Senator's theory; and because this theory seems to me to be based upon guesses and half-knowledge and some real facts; to claim recognition for a remedy thereon; to suggest to practical physicians that they may shape and bend their treatment of a disease on evidence supplied by such a medley group: I would deliver my challenge to it as an intruder into therapeutics,—with feeble voice, perhaps, but yet with voice of earnest protest.

First then, let me state, and at the same time bring into comparison for future use, the conclusions put forward in regard to the effects of salicin and salicylic acid in acute rheumatism, and these as stated by the advocates of the remedies respectively.

The effects following the use of salicin, as stated by its advocates,* agree in some of their features with the effects allowed for salicylic acid. At the same time, certain marked

* See Ziemssen's Cyclopædia of the Practice of Medicine. Vol. xvi. Appendix.

discrepancies are observed. Both remedies, it is said, reduce temperature and relieve pain, and fever and pain usually abate together. These are the earliest effects, and they are produced quickly. For, in most cases, the temperature becomes normal and pain ceases within 48 hours, and sometimes within 12 hours from the first dose. It is not disputed that within these limits of time the acid reduces temperature somewhat more rapidly than salicin, and relieves pain more quickly. But it is contended that the reduction of temperature and the relief of pain by salicin is more sure and abiding. Both remedies have to be given in nearly the same amount; the dose of each ranges from 10 to 25 or 30 grains. And in favour of salicin, its advocates say that it may be given every three or four hours, as against every hour or two hours for salicylic acid. Both remedies given at the very beginning of an attack may arrest the progress of the attack—like quinine in ague—the preference in this respect being claimed for salicin. Salicin is claimed to be better than the acid in very acute attacks. Salicin not being manufactured from carbolic acid (as salicylic acid is) nor liable to be contaminated with it, is free from the suspicion of producing those poisonous effects which are known to have sometimes followed the use of salicylic acid. And on the negative side—it is allowed that neither remedy has any power to influence the onset of, nor, being in progress, the course of any complications whether cardiac, or pulmonary, or of the brain.

Next, let me state Senator's theory. By this theory, if it be true, the alleged agreement, and also discrepancy in the effects just summarised might be accounted for; and the claims of salicin might be supported. Senator learns from chemistry that salicin very readily splits up under the influence of ferments, like synaptase and ptyalin, into glucose and a body named saligenin. He also learns that saligenin may be oxidised, out of the body, into salicylic acid. So he supposes that when salicin is swallowed its dissociation into glucose and saligenin (under the influence of ptyalin or other ferments) begins as soon as it enters the

stomach; that oxidation of saligenin into salicylic acid succeeds the dissociation, and takes place in the blood. And he states, as a fact established, that the salicylic acid so formed out of salicin is excreted in the urine partly as such and partly in the form of salicyluric acid.*

Upon this transformation, says Senator, the action of salicin depends (auf diesen umwandlungen beruht die wirkung des salicins.) The proof (beweis) of the transformation is a purple-violet reaction which salicylic acid and salicyluric acid each give with ferric chloride. Senator states that he has found this reaction in his own urine after taking 1·5 or 2 grammes of salicin, and within 20 minutes after taking the dose. Thus it may be supposed, at the tissues (where the effect is needed) both salicin and salicylic acid, though taken into the stomach in separate and different form, are the same body, salicylic acid. And the theory may be taken not only to so account for the concordance in the respective effects of the two remedies, but also to account for the discrepancy in these actions. For, it may be supposed, salicylic acid made out of salicin in the body is presented to the tissues *in statu nascenti*, almost at the instant of its formation, fresh and in its essence, and in its most impressive state. The effects of salicylic acid, given as such, may be quickly-wrought and striking; but if the supply be not quickly renewed the effects wear off too soon. The effects of salicylic acid, given as salicin, may be tardily-wrought and sober, but they are decisive and continuous, and renewal may be longer delayed.

The theory is essentially a chemical theory; it asserts that salicin undergoes chemical transformation into salicylic acid within the body. Wherefore its discussion and criticism must be, in the first place, from the chemical side. Is there any evidence that salicin can be and is converted in the body into salicylic acid?

* Berliner Klinische Wochenschrift. No. 14. 1877.

If there is evidence of the transformation, is this sufficient to bear out the contention of common utility based on such chemical identity? An adequate criticism and discussion of this question of chemical identity involves a detailed enquiry into the chemical relations between salicin and salicylic acid, and into the means and conditions we know of, existing in the body, whereby the conversion of one into the other may take place.

Salicin and salicylic acid may each claim organic origin. The former comes from the leaves and young bark of the willow and poplar and other trees. The latter comes from the winter green (Gaultheria), out of an oil prepared from this plant, and it can also be prepared from salicin itself. This first relationship of origin is important. The salicin we use in medicine is always prepared from the bark of the willow and other trees. The salicylic acid we use in medicine is prepared in a cheap and easy way from carbolic acid (phenol) by a process discovered by Kolbe in Germany. We mark here at the outset a difference rather than a relationship and identity. The salicylic acid we use in medicine, and given as such, may have the same chemical formula, and answer to the same colour-test as the salicylic acid made in the body out of salicin. But are they therefore the same and identical for Senator's purpose? In some respects they are certainly not the same. Phenol-salicylic acid (as I will term it on account of its source) sometimes carries with it, in spite of careful purification, poisonous or deleterious qualities which salicin-salicylic acid (in Senator's sense) has never been known to produce.* There are, as we have seen, marked differences in some of the constant effects of each; differences allowed by Senator. Can these discrepancies be explained by a reference to the nascent state? The nascent state has a reality and a fixed place in the science of the chemist; it has

* This might be an argument in favour of the clinical superiority of salicin; but an argument it would be of interest to follow up *after* the fact of transformation had been established.

no reality and no place here in regard to such a body as salicylic acid. We have no right to deal otherwise with what is furnished to medicine by another science, than would be allowed by the science to which it belongs; we have no right to ransack chemistry by picking out a fact here or a deduction there, nor to mould our plunder to fit our theory. We have no right to take the facts and deductions of chemistry for more or for less than chemists would take them to mean. The doctrine of the nascent state cannot serve any useful purpose here. We can understand how that hydrogen (for instance) may be in the nascent state. The molecule of hydrogen is complete; its bonds of union are fully satisfied, and disunion of its constituent atoms must precede combination with other bodies. This is the condition of hydrogen when free. The atom of hydrogen is incomplete, and its bond of union is unsatisfied and ready for whatsoever combination it may be able to effect. This is the condition of hydrogen in the nascent state. Thus:

$$\underbrace{H - \quad - H}_{\text{molecule.}} \qquad\qquad - H \qquad H -$$

atoms.

Now, salicylic acid is a compound molecule; it has, like hydrogen in the free state, all its atoms in union, and all its bonds fully satisfied by the union of the atoms amongst themselves In the act of its formation it never assumes a condition comparable to that of the separated atoms of hydrogen. There can be no such thing as the nascent state in regard to such a body as salicylic acid.

Let us look closely at the chemical relationships of salicin and salicylic acid, and enquire what these may have to do with oxidation—the process by which Senator says his transformation is brought about.

The chemical formula for salicin is $C_{13} H_{18} O_7$; that for salicylic acid is $C_7 H_6 O_3$. The two bodies agree in being made up of the same three elements; they differ in having different quantities of each of the three elements. Salicin may be represented as being made up of two things—glucose or grape—sugar, and saligenin.

It may be described more accurately, perhaps, as a compound of Saligenin and another body, which other body by taking up water becomes glucose, thus:

$$\underset{\text{Salicin.}}{C_{13}H_{18}O_7} = \underset{\text{Saligenin.}}{C_7H_8O_2} + C_6H_{10}O_5$$

$$C_6H_{10}O_5 + H_2O = \underset{\text{Glucose.}}{C_6H_{12}O_6}$$

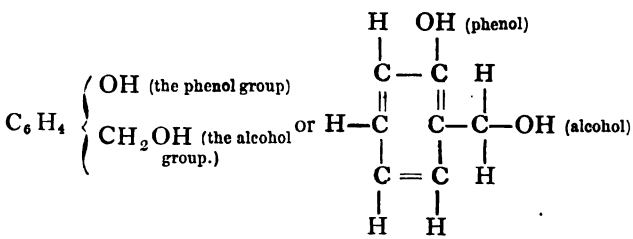
In presence of water salicin very readily yields glucose and saligenin; the action of saliva is enough to bring about the change. Many bodies are known, some of them ready-made in plants, having a similar constitution and liable to the same change. Wherefore chemists have called such bodies glucosides; salicin is said to be a glucoside of saligenin.

Senator supposes that salicin breaks up and yields glucose and saligenin as soon as it enters the stomach. The stomach is not the only place in the body, however, where Senator might suppose this conversion to take place. In the stomach there is ptyalin competent to effect the change, but there are also ferments in the intestine. In the small intestine the change would have to be effected in a more or less alkaline liquid; in the stomach it must be effected in presence of acids, and we know that out of the body saligenin becomes converted into a resinous body—saliretin—under the influence of dilute acids. Important considerations as to the conditions of acidity and alkalinity arise here, and should be decided before Senator's supposition can hold good. But we may, with reservation, admit the formation of saligenin out of salicin under the influence of ferments, either in the stomach or in the small intestine. Senator's theory really turns upon the fortunes of this body, saligenin.

Saligenin is commonly spoken of as an alcohol. It is true that it has a constitution and a place in organic chemistry which entitle it to be called an alcohol. Strictly, it is a glycophenol; it is a body intermediate in constitution and chemical behaviour, between

a glycol (an alcohol of the fatty group,) and a phenol (an alcohol of the aromatic group). This body suffers changes in oxidation which are relevant to the present enquiry; for it is in these changes that Senator's transformation of saligenin into salicylic acid (if it occurs through oxidation) must consist. And to supply an omission on Senator's part, let me speak of these changes somewhat fully, and take nothing for granted.

Saligenin is half an alcohol (true alcohol) and half a phenol, and the changes it suffers in oxidation, with which we are now concerned, occur in its alcohol portion.*



* The difference between a true alcohol and a true phenol depends upon the way in which the carbon atoms of their fundamental hydro-carbons are united. In a true alcohol, the bond between any two carbon-atoms is a single bond, and it is not necessary that every carbon-atom should be united with two other carbon-atoms. In a phenol, every alternate pair of carbon-atoms are united by a double bond, and each carbon-atom must be united with two others. When hydroxyl (OH) replaces hydrogen in the hydro-carbon of a true alcohol, it becomes united to a carbon-atom that is also united to hydrogen: in the case of a true phenol, hydroxyl becomes united to a carbon-atom that is not in union with any hydrogen at all. In an aromatic alcohol (to which group the phenols belong), a second hydro-carbon radical may be joined to the fundamental hydro-carbon, replacing a hydrogen-atom in the latter. Hydroxyl may replace a hydrogen-atom in this secondary hydro-carbon, and then it will be united to a carbon-atom which is also in union with hydrogen; hydroxyl is then in the alcohol place. And if a second molecule of hydroxyl should still be united with a carbon-atom of the primary hydro-carbon (not in union with hydrogen), this molecule of

Like ordinary (ethylic) alcohol, saligenin is the lowest term in a series of bodies derived from one another by oxidation. When ordinary alcohol is exposed, under suitable conditions, to the action of oxygen, some of its hydrogen is removed, and uniting with oxygen forms water. The remaining body, *acetic aldehyd*, differs from alcohol only in that it has two atoms less of hydrogen. When alcohol is still further oxidised, beyond the stage at which acetic aldehyd is formed, oxygen unites itself with the elements of the aldehyd to form a third body, *acetic acid*.

The series and the relations of its members may be thus expressed:—

$$C_2H_6O \text{ Alcohol.}$$
$$\text{Alcohol, } C_2H_6O - H_2 = C_2H_4O \text{ Aldehyd.}$$
$$\text{Aldehyd, } C_2H_4O + O = C_2H_4O_2 \text{ Acid.}$$

In organic chemistry there are many series like this, having each its alcohol, and its aldehyd, and its acid, and these related to one another—through loss of hydrogen and gain of oxygen—exactly in the same way. The alcohol saligenin has its aldehyd, formed by loss of hydrogen in oxidation. It is a yellow sweet-scented oil,

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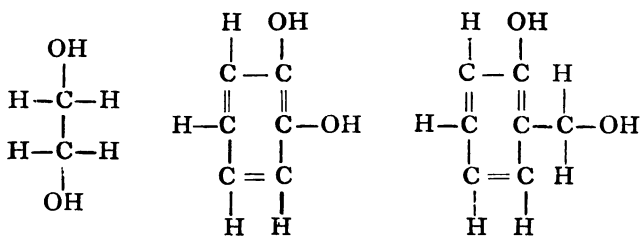
hydroxyl would be in the phenol place. Saligenin is such a body as would be formed by the union of one molecule of hydroxyl with a carbon-atom of the primary hydro-carbon, and the union of another molecule of hydroxyl with a carbon-atom of the secondary hydro-carbon: it is a diatomic alcohol with phenol characteristics besides. The following graphic formulæ will illustrate what has been said above.



Glycol. Phenol. Glyco-phenol. (Saligenin.)

called salicylol, and it is found ready-made in the flower-buds of the meadow-sweet (spiræa); its formula is $C_7H_6O_2$. In further oxidation salicylol takes up oxygen and becomes salicylic acid, the very body we know so well.

Salicylic acid is the acid of the series, and it is related to salicin through the intermediate bodies saligenin (the alcohol), and salicylol (the aldehyd). Thus:

$$\text{Salicin, } C_{13}H_{18}O_7 + H_2O = \underset{\text{Saligenin.}}{C_7H_8O_2} + \underset{\text{Glucose.}}{C_6H_{12}O_6}$$

$$\text{The alcohol} = C_6H_4 \begin{cases} OH \\ CH_2{*}.OH \end{cases} = \text{Saligenin.}$$

$$\text{The aldehyd} = C_6H_4 \begin{cases} OH \\ C.OH \end{cases} = \text{Salicylol.}$$

$$\text{The acid} = C_6H_4 \begin{cases} O\,H \\ C\,O{*}.OH \end{cases} = \text{Salicylic acid.}$$

Senator supposes that saligenin becomes converted into salicylic acid by oxidation (as he expressly says) within the body.

The relations just now so fully set forth, point out the mode and route by which this conversion should be effected. Whatever may be the means of conversion, the mode and route of conversion, whether it takes place in or out of the body, should be the same. The mode is by abstraction of hydrogen and addition of oxygen, and the route lies always through alcohol and aldehyd to acid. At least, our knowledge of the means by which the conversion of salicin into salicylic acid may be effected out of the body allows us to say so much. Thus, saligenin is converted into the aldehyd, salicylol, by means of the oxidising agent chromic acid and moderate heat. Salicylol is converted into salicylic acid by means of cupric oxide and a boiling heat. Other oxidising agents are of more or less avail. Salicylol is partially oxidised to salicylic acid

* It is to be observed that the hydrogen in the alcohol group which suffers displacement in oxidation, is easily displaced; and that the oxygen which replaces this hydrogen, and is found in the acid, replaces it easily.

by means of potassium dichromate and sulphuric acid. And potassium hydrate avails for the conversion of salicin into salicylic acid; but the potassium hydrate must be in excess and added continually, else salicylol alone is formed. The only process that I can find anywhere described, which is not unequivocally a process of oxidation by chemical agents, is one devised by a Russian chemist, Tichenowitsch. This chemist has converted salicin into salicylic acid by a current from a Bunsen's battery of four hundred pairs; and he says that in the process salicin becomes glucose and saligenin, this latter becomes salicylol, and salicylol becomes finally salicylic acid.

If there exist within the body means and conditions whereby the conversion may be effected, these no doubt will be found in processes of oxidation; and the route of conversion within the body may be expected to lie through alcohol and aldehyd to acid. But what knowledge we have of any conditions existing within the body for the required oxidation, forbid us to allow that—even granting oxidising agents—the conversion could be effected readily or fully, even if it be effected at all. Our knowledge of the whole subject of oxidation within the living body in a state of health is not much; our knowledge of it in the living body in a state of disease is nil. What, then, our knowledge may enable us to say cannot be applied to account for what should happen in disease, without much caution and great reservation.

When we seek within the body for means and conditions for oxidation, whereby this assumed change of saligenin into salicylic acid may be brought about, we turn first, and naturally, to the hœmoglobin of the red corpuscles of the blood as the source of oxidation most likely to avail us. But when we look to it for help we find only that it *might*, by bare possibility, be the effectual agent in the change; it is at best, a broken reed and a doubtful basis on which to build.

We know that hœmoglobin is the great carrier of oxygen to the tissues, and that it carries its oxygen in a loose sort of combination,

so that its oxygen can be disconnected without alteration in the composition of the molecule of hœmoglobin. We know also something about the conditions of disconnection of oxygen from hœmoglobin. In the case of blood withdrawn from and outside the living body, the loosely-held oxygen may be disconnected by certain gases, and by reducing agents like ferrous sulphate. And in the case of blood inside the living body, the oxygen may be, and is withdrawn by living tissues. On the other hand, our knowledge goes to shew that oxidation takes place in the tissues and not in the blood at all.* When oxygen is disconnected from hœmoglobin by the tissues, it gets fixed in them; and there, in the tissues, helps to form compounds whose subsequent destruction gives rise to carbonic acid. Nor does the production of carbonic acid depend directly on the absorption of oxygen by a tissue, for muscle produces carbonic acid in an atmosphere of hydrogen. And then we have this, for our present purpose, most suggestive fact: "many easily oxidised substances, *such as pyrogallic acid,* pass largely through the blood of a living animal without being oxidised; the organic acids, such as citric, even in combination with alkaline bases, are only partially oxidised; when administered as acids and not as salts they are hardly oxidised at all."† It would, then, seem as if the oxygen of hœmoglobin, loosely-held though it be, had the tissues for its only destination, there in the tissues to suffer disconnection, and to take part in constructive and destructive processes under conditions we do not know. It would seem as if substances which are denied an entrance into the tissues, easily oxidised and easily diffusible though they be, fail to find in their transit through the blood conditions enabling them to disconnect oxygen from hœmoglobin, and to use the oxygen for their own conversion into other forms. Saligenin in solution may be easily oxidised—though a glyco-phenol, it behaves to oxidising agents

* Text-book of Physiology, Dr. M. Foster, pp. 247 and 326.

† Ibid.

like a primary alcohol—but other equally easily oxidised substances fail to get an entrance into the tissues, and fail to get oxidised on their way to the tissues. If hœmoglobin is to oxidise rapidly large quantities of saligenin or salicylol—and these are conditions in Senator's theory,—and this in addition to its own proper work, oxyhœmoglobin should be proved to have other properties, and to be amenable to other conditions than physiology and chemistry have as yet made out. At least, the work to be done is difficult, and the conditions of the doing are complex and unknown; and if the work should ever be done, it could hardly be without grave interference with lawful physiological work.

But may there not be other means and conditions in the blood whereby the required transformation might be effected? The transformations of waste products, doubtless through oxidation in part, might be thought, at first sight, to supply evidence of oxidation normally performed in the blood. But there is no evidence that any conditions exist in the blood whereby these transformations may be brought about in it; on the contrary there is every reason to believe that they are effected in tissues and not in the blood.

I examined some blood withdrawn from a patient suffering from acute rheumatism and pericarditis, who had taken large doses of salicin during the previons sixty hours. The blood was extracted with ether, and treated with concentrated sulphuric acid; the red colour reaction characterising salicin was readily produced; no effect whatever was produced when the ferric chloride test was applied.

And where else in the body shall we look for such means and conditions as may supply the needs of Senator's theory? The teachings of modern physiology point with some assurance to the tissues and organs of the body, as the seat of those chemical changes and transformations (involving oxidations,) which we designate by the general term metabolic. Have we any ground for supposing that Senator's transformation may be referred to the

tissues or organs of the body, and to metabolic changes going on in them?

Here, in this region, we shall certainly meet with many theories that are only guesses, and with many facts that are only half-known facts—some of them hotly contested. Here, if anywhere, it might be forbidden to go in search of material wherewith to frame a theory; here, if anywhere, it might be forbidden to go in search of evidence to refute the theory. But there is a reason why this region should not be left out in our discussion of Senator's theory. Though we may find, with our present knowledge, no evidence whatever that Senator's transformation can take place, the fact is asserted by Senator that after ingestion of salicin, salicylic acid is actually excreted in the urine. If this fact be true and sufficient, we need not revise our chemistry and physiology. We may have to admit that our little knowledge, sound enough so far as it goes, is only not enough; we may have to admit the existence within the body of means and conditions that have not yet been fully grasped. In presence of this fact, if it be true or incapable of another interpretation than that which Senator gives to it, we cannot very well refuse to credit something and some conditions in the body with ability to do what, in spite of present knowledge, is done. To this we add—is done in part if done at all.

Senator starts with saligenin in the stomach, he ends with a test for the presence of salicylic acid in the urine. He contends that, therefore, somewhere in the interval between the stomach and the urine his transformation must take place. We have found no evidence that the blood is the seat of the change; the tissues and organs of the body assert themselves as the next and only remaining possible seats of the change.

Before entering upon this region of enquiry, the fact of excretion of salicylic acid must be considered. Let me then first discuss the crucial test which Senator takes as the proof of the change. In discussing it we may, perhaps, find a guide amidst the perplexities and uncertainties which await us.

Salicin gives no colour-reaction with ferric chloride; saligenin gives a deep indigo-blue reaction, and salicylic acid gives a purple-violet reaction with the same reagent. There is no mistake about these tests; the absence of all coloration in the one case, and the depth and contrast of hue in the other cases, are striking and distinct. Senator takes salicin, and then, testing his urine with ferric chloride, finds the violet reaction. The conclusion is obvious, and might, perhaps, be admitted as valid but for this other fact: salicylol, the aldehyd, strikes with ferric chloride the same purple-violet colour that salicylic acid strikes. The test cannot be a proof of the presence of salicylic acid in the urine. And the force of this failure in the test, as a proof of Senator's position, becomes more clear when we reflect on what this body salicylol really is. This aldehyd, sometimes called salicilous acid, has acid properties; it decomposes carbonates and yields crystallisable salts; with potassium and sodium it forms acid salts which are stable and permanent when dry; the acid salt of potassium crystallises in a form similar to that in which salicylic acid crystallises. This body then, in one form or another, might appear as easily as salicylic acid in the urine. And to complete the matter, we have no need to fall back on saligenin for the origin and source of this salicylol. We might suppose that salicylol was derived from salicin indeed, but by conversion of this into helicin ($C_{18} H_{16} O_7$) instead of saligenin and glucose. This conversion would require no more than what is required in order to the conversion of saligenin into salicylol—the loss of two atoms of hydrogen. And then, finally, we might suppose that helicin became changed into glucose and salicylol ($C_7 H_6 O_2$) under the influence of the same ferments that convert salicin into glucose and saligenin. Although we should have warrant for this in the known transformations of helicin, yet to advance this view in criticism of Senator, would be only to replace one supposition by another.

A third body, salicyluric acid,—a body having intimate relations with salicylic acid—also gives the purple-violet reaction with ferric

chloride. This Senator knows, for he says that the salicylic acid formed out of salicin is excreted in the urine partly as such, and partly as salicyluric acid. We cannot admit that the ferric chloride reaction is sufficient proof of the excretion of this body—salicyluric acid—after the ingestion of salicin, any more than it is proof of the excretion of salicylic acid. But upon the chemistry of this body and some other proofs of its excretion in the urine, hangs the only shred of evidence that can be found in justification of Senator's theory—and this scrap of evidence is only indirect, and insufficient to support the theory.

What then is this body, salicyluric acid?

When salicylic acid is ingested, part of it finds its way into the urine unaltered, but part becomes converted into a complex substance compounded of salicylic acid, and of a constituent of the glycocholic acid of the bile, glycocine: this complex substance is salicyluric acid. The process by which this colligation of glycocine and salicylic acid comes about, is analogous to that well-known process by which benzoic acid becomes converted into hippuric acid; and salicyluric acid and hippuric acid are both excreted in the urine. The process, as for salicyluric acid, may be thus represented:—

$$\underset{\text{Glycocine.}}{C_2H_5NO_2} + \underset{\text{Salicylic acid.}}{C_7H_6O_3} = \underset{\text{Salicyluric acid.}}{C_2H_4(C_7H_5O_2)NO_2} + H_2O$$

The union appears to result from the ejection of water (H_2O) from the two bodies; similarly, under hydrolytic influences, the colligated acid body resulting from their union, becomes split up into glycocine and salicylic acid by assumption of water. This mode of regarding the process may be made intelligible by altering the formulæ, thus:

$$\underset{\text{Salicylic acid.}}{C_6H_4\,(OH)\,CO.\underbrace{\mathit{OH}\qquad\mathit{H}}_{\text{removed or taken up.}}.HN.CH_2.COOH.}$$

Glycocine.

Salicyluric acid would thus appear to require for its formation, that salicylic acid should be pre-formed in the body. It is certainly difficult to see how salicyluric acid could be formed easily, and in accord with chemical notions, out of saligenin or salicylol.

Consideration of the following formulæ will make this statement clear.—

(1.) C_6H_4 (OH) CH_2OH $\quad$ HHN CH_2 COOH.

In this case we suppose salicyluric acid to be formed out of saligenin. It will be observed that here the group H_3 has to be removed.

(2.) $C_6H_4(OH)COH$ $\quad$ H HN CH_2COOH.

Here, we suppose the action to occur between glycocine and salicylol, and in this case the group H_2 has to be removed. And this seems to require the admission that the existence of salicyluric acid in the urine is evidence of the pre-formation of salicylic acid somewhere in the body, when salicin and not salicylic acid is ingested. So far Senator would seem to have indirect evidence of his transformation; but it is not evidence that he himself has adduced. Is this substance salicyluric acid actually excreted in urine after ingestion of salicin? Now, we have dismissed the colour-reaction as the proof of the formation of either salicylic acid or salicyluric acid in the body. Have we any other evidence that the latter, at least, of these substances is excreted in the urine? And if so, what is it worth?

After ingestion of salicin, salicylol and salicylic acid are found in the urine—so say Laveran and Millon.* These observers state that they have not been able to find any means of accurately estimating the amount of each in respect of the quantity of salicin ingested; that the violet coloration however is invariably produced; that they have obtained crystals of salicylic acid from the urine. Ranke, again,† gives the results of two experiments he made to determine in what forms salicin is excreted in the urine. He

* Ann. Ch. Phy. [3] xiii. 145. † I. pr. Chem. lvi. 1.

prepared an ethereal extract of the urine, and in this, he says, he found salicylol, and by crystallisation, an acid body "which shewed itself to be salicylic acid." No grounds are given for this statement. In a second experiment Ranke found undecomposed salicin.* and crystals which might from their description have been crystals of saligenin, but which Ranke took to be salicylic acid.

I have myself made experiments with a view to determine what what is excreted in the urine after ingestion of salicin. I gave salicin to a patient suffering from a severe attack of acute rheumatism. For a period of twenty-four hours (the patient being almost saturated with salicin) the urine passed by this patient was collected; it measured 1300 cubic centimetres. An ethereal extract of one-half of this quantity of urine was prepared, and from this all the crystals it would yield were carefully removed. The crystalline extract weighed ·423 gramme, and consisted of an acid body, supposed to be salicylic acid along with salicyluric acid. The acids could not be separated one from another, nor therefore the amount of each be determined, as no good method of separation is known; but there was no doubt that some of the crystals contained nitrogen. Now it may be fairly computed that the patient had taken 120 grains of salicin in the period during which the 1300 c.c. of urine was passed. 120 grains of salicin might yield, if all of it was converted into saligenin and glucose and thence into acid, 50 grains of salicylic acid. How much salicylic acid this patient excreted we cannot tell. But we know that only about 12 grains of mixed acid crystals were recovered from a quantity of urine representing 50 grains of salicylic acid. And this 12 grains represents glycocine as well as acids.†

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* I have already referred to the fact that I found undecomposed salicin in the blood.

† Graham states that out of the body, 25 parts of salicin produce only 6 parts of aldehyd. (Elements of Organic Chemistry.)

We might be willing to allow, on this evidence, that the existence of salicyluric acid in the urine does imply the preformation of salicylic acid in the body; we might allow that the salicylic acid had been formed from salicin in the way that Senator assumes. But then, what becomes of Senator's theory of clinical common utility in chemical identity? It must certainly be shewn that the quantity of salicylic acid produced is enough to effect appreciable therapeutic results. And we have the fact, allowed even by Senator, that much larger doses of salicylic acid than of salicin are required to produce a remedial effect.

This discussion, into which we diverged when about to speak of tissues and organs as the possible seat of change from salicin to salicyluric acid, shall be our guide in the further consideration of our subject. The results arrived at may spare the necessity of a minute exploration of a country as yet nearly unknown, and so intricate, that an exploration in the present state of knowledge could promise no results. It will be enough if I refer to a localisation and possible mode of formation of salicyluric acid suggested by the discussion.

When hippuric acid is formed, the colligation between benzoic acid and glycocine almost certainly takes place in the liver; for, if measures are taken to prevent the passage of benzoic acid through the liver the acid appears in the urine as benzoic acid, and no hippuric acid is found in the urine at all. Glycocine does not exist as such in the liver. The conjugation between it and benzoic acid is not a mere union by dehydration of these two bodies ready prepared for union; active processes, such as may separate the glycocine from the cholic acid with which it is conjugated, are first required in order that the colligation between the glycocine and benzoic acid may come about. This points to activity of hepatic cells, and their participation in the process. And we shall not strain analogy if we suppose that the seat of the formation of salicyluric acid is also the liver, and that its production is due to activity of hepatic cells. Now the route into the systemic blood

—and therefore to the tissues affected in acute rheumatism—for all those things which might be formed out of salicin in the stomach or intestines, would be by absorption into the veins of these organs and thence through the portal system. But there is no evidence to shew that once in the blood a further conversion to salicylic acid could there take place. And if some salicin, or saligenin, or salicylol should be found in systemic blood, this might be overplus which had escaped transformation and conjugation in its passage through the liver. So that we may well believe that some salicin and all the products derived from it by the action of ferments in the stomach or intestines, saligenin or salicylol—(as made out of helicin)—must pass through the portal system into the liver, and there come into direct relations with the hepatic cells. And, further, we may well believe that here in the liver, and by the agency of hepatic cells, the transformation occurs whereby salicylic acid is formed (out of saligenin or salicylol,) and this is conjugated with glycocine to form salicyluric acid. For in the liver, and by the agency of hepatic cells, hydrolytic decomposition of glycocholic acid into glycocine and cholic acid takes place, and cholic acid dissolves red corpuscles and sets oxygen free; or, some oxyhæmoglobin (even venous blood is not all de-oxygenated) may be carried to the liver from the spleen along with the bile-pigment which there is reason to believe is formed partly in the spleen and undergoes further transformation in the hepatic cells. And saligenin, or preferably salicylol in the form of its potassium or sodium salt,* being present there, it is easy to conceive how salicylic acid might first be formed; and glycocine being also present there, it is easy to conceive how the final step might be the formation of salicyluric

* Sodium salicylol, for instance, is not a salt in which the base has the usual relations with the acid. In this case the sodium appears to replace the hydrogen-atom in the phenol group of salicylol, thus:—

$$C_6 \cdot H_4 \begin{cases} OH \text{ (phenol group.)} \\ C.\ OH \end{cases} \qquad C_6\ H_4 \begin{cases} O\ Na \\ C.\ O\ H \end{cases}$$

Salicylol. Sodium salicylol (salicylicite.)

acid. Thus the suggestion might be allowed to have some ground, that the salicylic acid which is excreted as salicyluric acid (and along with it) may be formed in the liver at the same time when, and in the same place where, conjugation between it and glycocine occurs.

But then what becomes of the theory that the therapeutic action of salicin must be fundamentally the same as that of salicylic acid? Where is the evidence that shall give us the right to say that salicin, ingested as such, is at the tissues affected in acute rheumatism in the form of salicylic acid—much less salicylic acid in the nascent state—and there and thus no less in quality and power and action than what we know the salicylic acid made outside the body and ingested as such to be?

The theory before us has also a clinical or therapeutic significance. I proceed now to discuss the relations of salicin and salicylic acid from a clinical point of view.

The circumstances under which this theory is put forward are peculiar, and they are exceptional in the history of therapeutics. A criticism of the theory from the clinical side has therefore peculiar features special to the circumstances. Here we have a remedy tried by experience, and that not a narrow experience; a remedy found to succeed, and that on authority almost universal—salicylic acid, to wit. Here we have also another remedy, salicin, with claims contested, and a reputation only in the opinion of the few. A theory is put forward which may fairly be regarded as a plea for salicin, and by one who advocates already its therapeutic claims;* and the theory accounts for the efficacy and special effects of salicin by supposing that salicin becomes converted into

* When the theory was first put forward it would seem Senator had not tried salicin in acute rheumatism, and only in so far as temperature was concerned had he observed its effects in other diseases. In the article in Ziemssen's Cyclopædia of Medicine, however, Senator is quoted as an advocate of salicin in acute rheumatism, and this article has Senator's own approval and endorsement.

salicylic acid. The nature of the criticism of a theory put forward under such circumstances will depend very much upon whether or not we are advocates of the remedial claims of salicin. As advocates of salicin we might say that the theory—incomplete though it be, and not yet fully supported by evidence, because this must as yet be based on imperfect knowledge—is sound so far as it goes, and is already fully justified by the test of experience. And then, those who contest the effects of salicin might retort that its advocates argued as if they said, "This body can cure this disease because that other body cures the same disease; and this body becomes that other body in the organism:" and the questions would be pertinent—Is experience, then, not to be the test of truth? or must we allow experience to test by proxy? Again, if the remedial effects of salicin be allowed, Senator has only to prove chemical identity in order to account for an admitted agreement in effects. Still, if it could be shown that salicin is in the body the same thing chemically as salicylic acid, it would by no means follow that all the effects allowed to salicylic acid should be transferred to and allowed to salicin.* If it could be shewn that salicin did become salicylic acid in the body, we could only *expect* of it remedial effects equal or similar to those of salicylic acid; we should still have to shew correspondence between the admitted effects of the one and those inferred for the other, even though we might be advocates] for salicin. We should still require the test of experience to be applied anew, and should have to await the further verification arrived at by methods adapted to the newer conditions. And if we are not advocates of salicin—if we have never used it at all—this would be very much the course we should have to adopt in regard to the theory. We should still have to put the theory to the test of experience, and await the verdict of experience upon it.

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* We have alluded to the difference between salicin-salicylic acid and phenol-salicylic acid, page 21.

Wherefore, first noting some obvious criticisms arising out of what has been discussed in the former part of this paper, I will adduce what evidence I may be able to find in my own experience to show that salicin is not therapeutically (as a remedy for acute rheumatism) identical with salicylic acid, and therefore can have no common utility with it. The agreement between the effects of salicin (as stated by its advocates) and those of salicylic acid is not sufficiently close to support the theory of chemical identity. Similarity in effects might certainly be expected from two medicinal substances having an identical chemical character. Instead, we note here great disparity in effects. It has been sought to account for this, it is true, by a reference to the nascent state—a fiction in the case we have already disposed of. Nor can the difference between salicin-salicylic acid and phenol-salicylic acid, due to source, be held to account for the marked differences in effects admitted by the advocates of salicin. As we shewed, identity due to source was identity with a difference—say, isomerism—but the difference now is one which no isomerism in any of its forms could account for. When Senator says that the action of salicin must be fundamentally the same as that of salicylic acid, he could hardly have had in mind the action of these remedies in rheumatism; for here is no pathology sufficiently exact to work upon; the pathology of the disease is as yet but theory. Effects of remedies here must be effects on symptoms, and these in rheumatism a very shifting sand. Much more indeed is needed to establish such a community as would be agreement, in a scientific sense, for acute rheumatism, than a bare statement of results arrived at by those who advocate a remedy on one side or those who do not on the other. It is not enough to allow that both remedies reduce temperature; nor enough to allow that under the use of both remedies pain and swelling subside in affected joints. The effects upon all the symptoms of the disease must bear resemblance when each remedy is compared with the other for similar cases, for rapidity of action,—for all those circumstances, in short, which constitute

for us two like cases of rheumatism, and are our gauge of the successful cure of the disease. It is notorious how that for acute rheumatism very much the same statements of demeanour, both of patient and of disease, come out for cases treated in one way as for cases treated in another and different way. Of all diseases acute rheumatism is the one that will least bear, perhaps, a reference to similarity in its comportment under different remedies in different cases, and that most needs comparison of the effects of its remedies to be guarded by strict assurances against error. To one such assurance against error, that of comparison of the effects of salicin and salicylic acid in the same cases, it occurred to me to appeal. And having used both remedies in the same cases, and noted the results, I am able to bring forward evidence of the failure of Senator's theory, guarded in this way against error.

Already, when Senator's theory first came to my knowledge, experience of the two remedies had taught me to prefer the one and reject the other. But then I made comparative trials of the remedies in the same patients. I gave salicin, in proper dose and form, for some time to patients suffering from severe attacks of acute rheumatism, and I saw little or no results beyond the single result it is admitted to give more tardily than salicylic acid—reduction of temperature. During the exhibition of salicin the urine gave the violet re-action with ferric chloride; then, after discontinuing the salicin, and finding the urine no longer gave the violet re-action, I gave to the same patients salicylic acid, also in proper dose and form, and I saw immediate and brilliant results. This experiment I have had the opportunity to repeat in a large number of cases during the past two years and more, and the results have been always much the same. Let me describe these two cases, selected from many others, shewing results which have led to a strong belief that Senator has erred, and that clinical experience in favour of salicylic acid is right.

Case 1. The patient was a married woman, aged 22. The attack was recent. The rheumatism was polyarticular, with the

usual concomitants, profuse sweating, pyrexia, &c. The bowels were first effectually cleared, and then treatment by salicin commenced.

Period i.—72 Hours. Salicin was taken in 15 grain doses every three hours, and during this period 360 grains were taken. The temperature rose during the first 24 hours and reached 104° (with pulse of 140); thence temperature decreased, and at the end of the period was 101° (with pulse of 108). No effect was produced on the pain, swelling of joints or other symptoms; indeed, more joints had become affected. Some epigastric pain, soreness of mouth and thirst were complained of, but at the end of the period these had passed off.

Period ii.—45 Hours. Salicin was taken in 15 grain doses every hour for 6 hours; thence in the same dose every 3 hours; in all 270 grains. Temperature rose 1° during the 6 hours, (pulse 106), and then decreased to 100°, (pulse 108) at the end of the period. Pain and all other symptoms, except the sweating, were as bad as ever at the end of the period; the tongue was brown, and the urine scanty, (no urine at all was passed for 24 hours from the beginning of the period). Some ringing in the ears occurred after the first 90 grains had been taken.

The patient had now been treated with salicin for 5 days. No improvement had taken place; indeed, the patient was rather worse than better. It was deemed hazardous to continue the salicin.

Period iii.—All medicine was discontinued for 24 hours. At the end of that time the patient was in much the same state, except that other joints had become affected.

Period iv.—Then I gave trimethylamine, a remedy I had formerly made much use of, for a period of 72 hours. Some relief was experienced, (although the medicine was not pure).

Period v.—72 hours. No medicine at all was taken.

The patient had not as yet been free from pain; the tem-

perature was now over 100° (it had been 101·2° a few hours before). There was no decided improvement in the symptoms, other than temperature, and the patient was still sweating profusely.

Period vi.—72 hours. Salicylic acid was taken in 15 grain doses every 3 hours. In less than 24 hours there was marked diminution in pain and in all symptoms. The clinical record cannot reproduce the changed appearance in the patient that struck forcibly all those who had been watching the case. In 60 hours all pain had ceased entirely, and it did not again recur. The temperature then was 98°.

Period vii.—48 hours. Salicylic acid was continued in 15 grain doses every 4 hours. At the end of the period the patient had no pain anywhere, she could move the limbs freely without pain, (the joints were only stiff), and was entirely free from all other symptoms of the attack. From this time, through a convalescence protracted by circumstances unconnected with the disease, recovery was uninterrupted.

Case II.—The patient was a single woman, aged 27. The attack was recent. The rheumatism was polyarticular, complicated by pericarditis. The patient had suffered from the disease three times before, the last attack two years before this. The bowels were effectually cleared and then the treatment began.

Period i.—For 144 hours the patient took salicin in 15 grain doses every 4 hours. At the end of the period the pain was still severe, much swelling and redness of some joints persisted, and the patient had no sleep the last night for the pain. The temperature was exactly the same as at the begining—101°. I make no mention of the pericarditis, since it is allowed on all hands that neither salicin nor salicylic acid have any effect on it.

Period ii.—For 72 hours salicylic acid was taken in 15 grain doses every four hours. In 24 hours there was very little pain

and the temperature was 99°. At the end of the period there was no pain or sweating or fever, and none recurred.

Some ten days after this, the patient having been up and going on well, a relapse occurred. This was speedily controlled by salicylic acid.

This clinical evidence confirms the doubtful character of Senator's speculation; it completes the proof that the theory has no sufficient foundation in fact to entitle it to a place in therapeutics.

On Sunstroke.

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Although the number of deaths from sunstroke in the United Kingdom forms but a small item in the Registrar General's returns, and although such an occurrence as an outbreak of sunstroke is practically unknown except in very warm or tropical regions, yet scarcely a summer passes without our reading, or hearing of, and perhaps seeing occasional cases of this malady during the hot months.

I ought to state *in limine,* that my experience of this disease has been a limited one as regards the acute attack, but I have had many opportunities of seeing and of dealing with its after consequences.

I have roughly put together a few remarks drawn partly from the history of such cases as I have met, and partly from the experience of medical officers of the Indian service. I do not think I have anything new to say; but at all events it may serve to ventilate the subject and elicit some discussion.

For the sake of brevity and with a view to some arrangement, and at the risk of being considered elementary, I will confine

myself to a brief reference to one or two remarkable outbreaks of the disease; to a consideration of its varieties and sequelæ; and to a few observations regarding its pathology and treatment.

I need scarcely say that we must look principally to the annals of Indian and American medical experience for the most reliable and instructive accounts of this formidable and fatal malady. As regards the former, were you to take the trouble to wade through the literature on this subject, you would find a sad record of the destruction of human life from this cause—sacrificed on some occasions it is true unavoidably, and in the heroic defence of the British Crown—but in others caused undoubtedly by official ignorance and obstinacy perversely exercised in direct opposition to medical advice and counsel.

Perhaps one of the best descriptions we have of an outbreak of this disease is to be found in the report of the Army Medical Department for the year 1859, by the late Dr. Barclay, Surgeon of the 43rd Light Infantry. During the eventful period of the Indian Mutiny in 1857, this distinguished regiment marched nearly across the entire Madras Presidency to Jubbulpore; the march occupied over four months, extended over a distance of 969 miles, and was performed entirely on foot. There were no railways in India in those days. In the endeavour to reach the scene of action in the north of India, the march was a forced one, and the effect upon the men naturally was very great; they became exhausted, worn, and emaciated. On their arrival at Jubbulpore it was anticipated that the regiment would have enjoyed a good long rest; but no sooner had they reached their quarters than peremptory orders arrived directing the commanding officer to march to Calpee, a distance of 160 miles, with all the speed possible. Up to this time the cases of sunstroke had been few. The heat although great had not been overwhelming. But now in the month of April these already exhausted men had to start once more and push their way as best they could. At one point in this memorable march a place called *Nagode*, Dr. Barclay describes the

heat as insufferable; the regiment was encamped in a valley with precipitous rocky sides. The heat in the tents during the day varied from 115 to 127 degrees of Fahrenheit, while even at midnight it seldom fell below 105: even the natives could not bear this, and many were struck down and died of sunstroke within an hour. As for the European soldiers, they were carried into the hospital tents at every hour of the day and night. On the march between Nagode and Banda, a distance of 100 miles, the regiment lost two officers and nineteen men, but the general condition of the men was such that Dr. Barclay states there was scarcely a man who was not reduced in strength to the level of a child.

This is an instance taken out of a great many others, which exhibits the liability of men who have undergone *great fatigue*—to the evil effects of long continued exposure to a high temperature.

But fatigue is not the only cause which seems to give rise to an outbreak of this disease. Over-crowding in a vitiated atmosphere combined with a high temperature, is a frequent cause. This was especially remarked by Professor Longmore, during an outbreak in the 19th regiment, at Barrackpore, in 1858.

A peculiar stillness and closeness of the atmosphere, combined with a temperature ranging beyond 98° Fahr., is almost certain to produce cases of insolation.

I am acquainted with an army medical officer of ability and experience, who was in the habit on leaving the mess room after dinner, of sniffing the air outside the door, and if he found this peculiar closeness he always went to the *hospital* instead of to his own quarters, feeling certain that cases of insolation would be brought to him, and he was seldom mistaken; and Dr. Barclay noticed, during the march I have referred to, that on the occurrence of a thunder-storm after sultry close heat, cases of solar apoplexy *ceased*,—no doubt owing not only to the fall of temperature, but also the alterations in the electric and barometric conditions of the atmosphere.

Dr. Staples of the 19th Regiment also states, with regard to an

outbreak at Nowshera, on the Indus, that on the 30th June, 1867 between 5 o'clock and 10 p.m., during very still and close weather, and when the heat may be said to have reached its greatest point of intensity, no less than 28 men were brought to hospital with all the *premonitory* symptoms of insolation—and so intense were their sufferings, that the most sanguine could not have hoped for anything better than a large proportion of attacks of the graver disease; but about 10.30 a severe land storm came on which had the effect of reducing the temperature from 105 to 95 Fahr., and in two or three hours these men had quite recovered.

A still and moist atmosphere, combined with high temperature, frequently induces an outbreak of insolation. Mr. Naylor, of the Indian army, observed in reference to sunstroke at Jhansi that although the thermometer at noon ranged from 110 to 120 Fahr. with bright sun, it was not upon such days that cases of sunstroke were most frequent, but rather during cloudy moist weather with little movement of air; and I can bear this out from my recollection of tropical service in the West Indies, that affections attributable to solar influence were less frequent during bright weather without clouds, with the sea breeze blowing, than in sultry weather without much movement of air, although the thermometer marked a higher degree of temperature in the former than in the latter, and for this reason, sunstroke is rare in the West India Islands, although on the continent of America within the same latitude it is frequent and fatal.

So much for some of the general causes. As regards special causes as affecting individuals, I may observe that it attacks the young, strong, and active, and specially those newly arrived in tropical countries. Fat men are particularly liable to it. My friend, Dr. Crawford, observed this fact in an outbreak of the disease in the 51st Regiment, at Rangoon. Others have made the same observation; and it would appear that this condition of obesity favours the generation and retention of animal heat.

There is but little doubt that persons addicted to the use of

alcoholic stimulants are more liable to attacks of sunstroke than the temperate. This is the opinion of every observer I am acquainted with, except one. Sir Ranald Martin, Morehead, Taylor, Maclean,—all regard intemperance as a predisposing cause. But Dr. Staples, late of the 19th Regiment, who I know to be a very careful observer, declares, in reference to a very severe outbreak of sunstroke at Nowshera, already alluded to, that no case occurred among the notoriously intemperate, or those who had been previously in hospital for delirium tremens or other effects of alcoholism; while one case occurred in a man of remarkably abstemious habits. Sir Charles Napier, however, in his account of his own case when serving in Scinde, characteristically says, "I was tumbled over with heat apoplexy, with forty-three other Europeans, they all died except myself within three hours. I do not drink!—that is the secret. The sun had no ally in the liquor amongst my brains."

Two varieties of this disease have generally been recognized,—the cardiac and the cerebro-spinal. Sir Joseph Fayrer, however, splits up the latter into two, and regarding the cardiac variety of Morehead and others as heat exhaustion attended with failure of the heart's action and syncope, he recognizes two other forms,—one a condition like shock, in which, from the direct action of heat on the brain and cord from exposure to the sun, the nervous centres are influenced, and death takes place from rapid failure of respiration and circulation. This Fayrer regards as true *Ictus solis;* the other form is Thermic, or Sun fever. The first of these is the variety frequently met with by medical officers in the field in India and other hot climates, when men have been engaged in active operations requiring protracted exposure to the sun's power. Large numbers of our troops perished in the defence of Lucknow from this cause. But sunstroke among European troops in India is now neither so frequent nor so fatal a disease as it formerly was, mainly owing to the fact that the soldier's dress and accoutrements are made on better principles than formerly: he is

no longer pinioned in the tight-fitting leather stock and heavy clothing of thirty years ago; and greater attention is paid to saving him as much as possible from solar exposure.

The third form of the disease is Sun fever, a condition of intense pyrexia arising from super-heating of the blood. The nervous centres become overwhelmed, not only by the constant and direct application of heated air to the entire surface of the body, but also by the incessant inspiration of the same, by which the blood circulating in the lungs is exposed to a temperature often much higher than its own, and that for a considerable time. For it appears that a necessary factor in the production of symptoms of insolation is, that there should be *prolonged* exposure to solar heat. Were such symptoms liable to be produced by exposure to *heat alone* for a short time, we should have cases of insolation occurring constantly in over-heated workshops and manufactories and foundries, and in the stoke holes of steam vessels.

I may digress for a moment to observe that according to the researches of Obernier, who has made experiments on animals exposed to a temperature of 40 C. = 104 Fahrenheit, such animals experience a rapid increase of body temperature; and he states that 77 per cent. of this increased heat is retained in the body unless certain regulators of heat show compensatory functional activity, this being brought about by increased action of the skin, increased respiratory movement, and augmented secretion from the kidneys. The animals exhibit increased cardiac action, and if the body heat reaches 44 C.=111 Fahrenheit, there are usually strong convulsions; they become unconscious; the body-heat increases, and death ensues. The post mortem appearances are substantially the same as have been observed in sunstroke. The right side of the heart is found distended with blood, and the lungs deeply congested.

Obernier confirms the pathology of sunstroke as generally received both in this country and in warm climates, observing that the necessary conditions for its production are *prolonged* exposure

to a high external temperature producing super-heating of the blood, and interference with evaporation from the surface of the body and from the lungs. That there may be, and probably is in addition, some direct action upon the nervous tissues, produced by protracted exposure to solar power, I have little doubt; but observations are wanting upon this point.

As regards morbid appearances, the prominent lesion is intense and extensive pulmonary congestion. My friend Dr. Todd, late of the 99th Regiment, has informed me, however, that he has met with fatal cases of sunstroke in which during life there was intense coma, but with easy respiration, attended however with bloody urine, the morbid appearances being intense cerebral congestion, a similar condition of the kidneys, while the lungs appear to have escaped.

While it is true, I think, that the prominent lesion in this disease is pulmonary congestion, the other internal organs exhibit more or less similar appearances. The liver, spleen, and kidneys are found deeply congested; the brain and membranes also exhibit the same phenomena; the sinuses are gorged with blood, and in some cases there is evidence of meningitis. The brain substance is deeply congested, and there may in some cases exist slight extravasations. These morbid appearances in the head gave rise to the hypothesis that insolation was an apoplexy, hence the name solar or heat-apoplexy. Indeed, the nomenclature of the disease exhibits in a striking manner the various pathological theories which have been entertained regarding it.

Ictus solis.	Sun fever.
Calenture.	Heat fever.
Heat apoplexy.	Heat apoplexy.
Coup de soliel.	Heat asphyxia.
Erethismus Tropicus.	Loo marna.

As regards the circulatory system, the blood is found dark and fluid, as in cases of death from sudden shock; patches of petechial character are observed on the surface, and the heart is found firmly

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contracted. This Sir Joseph Fayrer attributes to anti-mortem coagulation of the cardiac myosin; and Dr. Wood, of Philadelphia, adopts the view that the rigidity of the heart depends on the coagulation of the muscle-plasma, which occurs at 115° Fahrenheit,—a degree of body-heat almost attained in insolation; and he considers the cases of sudden cardiac deaths in India may be due to this coagulation of myosin. If, however, this view were correct it would be difficult to account for recoveries which have taken place in cases of sunstroke where the temperature has reached 109·4 (recorded by Dr. Sevick, in the American Journal of Medical Science) and still more in Mr. Teale's remarkable case of recovery from special injuries, where the temperature ranged from 108 to 122 for a period of nine weeks.

As regards the symptoms of insolation. In the cardiac variety of Morehead, or heat-exhaustion of Fayrer, we have pallor, vertigo and fainting from failure of the heart's action,—a condition which we often see in a slight form in our badly-ventilated and overheated and crowded churches and public rooms, when weakly persons are suddenly attacked with syncope. The same thing in a different form frequently occurs during the hot weather at our camps at Aldershot, Colchester, and the Curragh, where men are exposed during the greater part of the day to the sun, and undergo at the same time fatiguing exercise. Cases of heat exhaustion are frequent under these circumstances, and sometimes we meet with the severer forms of the disease.

In the severer forms of sunstroke, whether we give it the name of cerebro-spinal insolation, or thermic fever, there are certain premonitory symptoms which seem to precede with tolerable regularity the onset of the more formidable symptoms.

For a long time before the occurrence of cases of sunstroke in a camp in a hot climate every one suffers more or less from attacks of Lichen tropicus or prickly heat; this is, rightly or wrongly, generally considered a safety-valve, and the sudden or gradual disappearance of the cutaneous eruption is regarded with suspicion;

its suppression indicates in fact an arrest of cutaneous activity; the skin becomes rough and dry, the bowels constipated, and there are very frequent calls to pass water. These symptoms generally usher in the attack, which in most cases is sudden—the patient either falls down insensible or becomes comatose while lying down at night; in some cases there is violent excitement and alarm, and a condition very similar to that observed in hydrophobia, very soon followed by total insensibility. There are often spasms of the muscles of deglutition, aggravated by attempts at swallowing.

When the symptoms are fully developed there is stertor, rapid breathing, contracted pupils, pale face, frequent sharp pulse, and burning heat of skin. In Dr. Barclay's case a few were attacked with convulsions, which generally attacked the upper extremities, but in some instances they extended over the whole of the voluntary muscles, and were of the most violent description. The nature of the convulsive attacks is remarkable. They are not strictly epileptiform—the body is in a state of rigour, with startings and jumpings from side to side, fulcra being made of the heels, buttocks, and occiput. These symptoms are attended with involuntary discharges from the bowels and bladder.

I believe I am correct in saying that there is no disease known in which body temperature rises to such intensity as in insolation, unless it be in such special cases of injury as Mr. Teale's case. So far as I know, however, his case stands alone. The body temperature in sunstroke ranges from 105 to 115 degrees Fahr. This latter temperature is the highest that I am acquainted with, and has been recorded by Staff-Surgeon Roch, in medical charge of H.M. Transport *The Golden Fleece*, when stationed in the Red Sea, during the Abyssinian expedition.

As regards diagnosis, I think there are but two conditions with which sunstroke is likely to be confounded, viz.—apoplexy and alcoholic intoxication. In both there is coma and stertor. In sunstroke, however, the pupils are contracted, and the conjunctivæ deeply injected—the pulse is quick and sharp, and we have the

unfailing guide of the thermometer which alone will be sufficient to indicate the nature of the case. I can quite understand that a case of sunstroke occurring in this country in the streets may be mistaken by an ignorant policeman for drunkenness, and that life might be sacrificed in this way for the want of medical advice, but the diagnosis would be at once established by any intelligent practitioner.

I will now pass briefly to the question of treatment.

In former days, when the lancet was the great resource in all acute diseases, nearly every case of sunstroke occurring in the tropics was bled freely and repeatedly—among them, the great Sir Charles Napier.

Bleeding, however, is very seldom resorted to now—a more enlightened pathology has condemned it.

In the milder forms of the disease, by placing the patient in the shade and dashing cold water upon the surface of the body, recovery soon takes place; but in the severer forms, where the patient is either struck down suddenly by the direct action of the sun's power, or where thermic fever becomes established by a slower process, the conditions are of the most dangerous character, and require incessant attention by the medical adviser. In the absence of any distinct pathological facts regarding tissue change in the nervous centres in this disease, and looking at the question clinically, we must assume, I think, that our patients' danger arises mainly from intense hyper-pyrexia. To subdue that,—to act on the vasomotor system, while at the same time the heart's power is maintained, should be our aim. Briefly, this is mainly done by cooling the external surface of the body. In every movement of troops in India now, in the hot season, arrangements are always made for giving the cold douche to men who may be attacked with sunstroke.

As soon, therefore, as the diagnosis is established cold should be applied either by affusions or the cold bath, while the temperature is carefully watched and noted. Very great benefit will be found

from the use of cold water enemata, as suggested by the late Dr. Parkes; and if the patient can swallow, draughts of ice-cold water; also by rubbing the surface with ice, and placing ice in the axillæ.

Latterly a mode of treatment has been introduced, namely, the hypodermic injection of quinine, which bids fair to occupy a prominent place in the therapeutics of insolation. I am given to understand that a case of sunstroke has recently been successfully treated in the Bristol Royal Infirmary in this way, and we shall no doubt have the details of it from some of the physicians present. So far as I am aware this treatment was initiated by Surgeon-Major Hall, and it has been followed up by many other medical officers in India. In the *Army Medical Report* for 1872, a case of this disease treated successfully by hypodermic injection of quinine is very graphically described by my friend Dr. O'Leary, of the Royal Horse Artillery. A man was exposed for some time to the sun in shifting baggage close to the Kistna River, on the line between the Madras and Bombay Presidencies. He was young and healthy. The train was about to start when he staggered up to the carriages set apart for the sick saying he was dying. The bell had rung—the man could not be left behind; he was placed in the carriage, two medical officers entered the carriage with him, some ice was procured, and a good supply of water. The cold douche was employed, ice applied to the nape of the neck, and friction to the limbs; but although the temperature was reduced to the natural standard the patient was making no rally, the heart's action was failing, the jaws were closed, and insensibility complete. Under these circumstances quinine was injected hypodermically—after the first injection, the pulse was felt to flicker, and when a second and third injection had been used, the man sat up and recognised those standing near; within half-an-hour he was able to feed himself, and the next day he was convalescent. This mode of treatment has been, as I have said, pursued by others in India with good effect. The quantity of quinine altogether used in Dr.

O'Leary's case was about five grains. It appears to me that this drug, when such brilliant success follows upon its use, as in this case, acts mainly as a stimulant through the nervous system to the failing powers of the heart.

As I do not wish to prolong my remarks, I will briefly say, as regards the sequelæ of sunstroke, that it has fallen to my lot to see very distressing consequences from this disease, consequences which have led me to the conclusion that very serious organic changes to nerve tissue have resulted from the original attack. For many years I was in the habit of meeting with the cases of men whose health had been completely wrecked from sunstroke; and I know few conditions of the nervous system more baffling or disheartening to the physician. Some of the results I have witnessed have been as follows:—Intense and persistent headache, which resisted every form of treatment; hernicrania, evidently of a neuralgic character; epileptic attacks, more or less severe; loss of memory; a fatuous condition of mind, attended with great bodily emaciation. I shall never forget the appearance presented by an officer who arrived at Netley from India, after having suffered from sunstroke. He was emaciated to a shadow, and was so weak mentally as to be incapable for many months of attending to the ordinary concerns of life.

Paralytic affections frequently follow sunstroke, principally evidenced by loss of power in the upper or lower extremities, and tremulousness in speech; but I cannot remember to have observed herniplegia to result from this affection.

One circumstance may be observed in persons who, to use a common expression, have ever suffered from a "touch of the sun:" they seek the shade on every possible occasion, even in an English summer, and dread the solar rays as a dangerous enemy, and never stir abroad at mid-day without the protecting ægis of a green-lined umbrella.

How far children are the subjects either of the milder or severer forms of sunstroke, even in our temperate climate, or to what

extent such attacks may lay the foundation of future meningeal or cerebral mischief, I am not prepared to say; but I do not doubt from what I have observed of the sun's power upon strong men, that there are many cases not only of permanent cerebral disease in early life owing to this cause, but also instances of acute formidable symptoms as clearly due to mid-day solar heat as those with which we are familiar in the story of the Shunamite woman's son.

In the discussion which followed the reading of Dr. Fyffe's paper, Dr. E. L. Fox commented on the varied forms both of the primary symptoms and the sequelæ, and mentioned the fact that mental sequelæ generally assume some form of mania. He related a case in which epilepsy and furious outbursts of temper were the permanent results from an attack. In commenting on the pathology of these cases, he mentioned that certain lesions have been observed in the cells of the middle layer of the grey matter of the convolutions; dilatation of vessels, small hœmorrhages pigmentation and atrophy of the nerve cells being the principal conditions recorded.

Dr. Shingleton Smith alluded to three cases which had been admitted to the Bristol Royal Infirmary on the hottest days of the hot summer of the previous year. One patient died in convulsions twenty minutes after admission, the temperature reaching 111° Fahrenheit just before death. Another patient, whose highest temperature was 109°, died six hours after admission. The third patient, whose temperature rose to 107°, recovered after the hypodermic injection of five grains of quinine.

January, 1877.

Acute Miliary Tuberculosis.

DR. J. GREIG SMITH.

SEVERAL specimens illustrating the beginnings of acute miliary tuberculosis taken from two patients who had recently died in the Infirmary were exhibited. They were interesting on the common grounds that both showed a sufficient cause for the existence of the disease; that in both the cause was found in a somewhat unusual, though not unknown position; and that, in each, though the lungs were almost free from miliary tubercles, there existed a condition of chronic phthisis. In one, the affection had arisen from scrofulous testicle, in the other from chronic gastric ulcer; while in both the chief crop of tubercles was in the peritoneum.

The first case was that of a woman, aged 28, who had suffered progressively from gastric ulcer, pyloric obstruction, subacute peritonitis and persistent diarrhœa. She showed no symptoms of either lung or brain mischief.

The necropsy revealed the following:—

In the *stomach*, about two inches from the pylorus, and facing each other were two hard cicatrices each the size of a shilling. For several inches all round there was enormous hypertrophy of the muscular and mucous coats of the stomach wall. This thickening was so great as to permit the passage only of a director through the pyloric orifice.

The *peritoneum* was everywhere thickly studded with tubercles mostly of large size, and some of them pigmented and depressed in the centre, and they were also freely scattered over the mucous membrane of the ileus and large bowel.

At the apex of one *lung* was a well protected cheesy nodule the size of two peas with about a dozen small miliary tubercles in its immediate proximity.

The *brain*, to the naked eye, appeared fairly healthy.

The other case was that of a man aged 33, admitted with scrofulous testicle. The morning after admission he became delirious; that same evening the delirium changed into coma, which gradually deepened till his death seven days afterwards.

At the necropsy the globus major of the left epididymis was found in a cheesy condition; his peritoneum was glistening all over with tubercles; in the brain were the ordinary appearances of basilar tubercular meningitis; while one lung showed a condition as nearly as possible similar to that of the woman—a small cheesy nodule at the apex surrounded by a few miliary tubercles, —both lungs being otherwise perfectly healthy.

Having called attention to the general belief as to the immediate origin of acute miliary tuberculosis in infection from caseated inflammatory products, Dr. Greig Smith traced anatomically the course of infection in these two cases, and specially pointed to the fact that each case had shown infection from a double focus, one from lung and gastric ulcer, the other from lung and epididymis, while in neither had the lung infection advanced to any extent.

Dr. Smith made the following remarks in connection with microscopic specimens and drawings laid before the meeting.

Cases of acute tuberculosis in which the cerebral meninges are but slightly affected, afford the best material for the study of the beginnings of the disease. The following is a summary of the appearances got from microscopic examinations of the cerebral vessels of nineteen cases of acute miliary tuberculosis, four of which—the most valuable ones—showed no naked eye lesion in the brain.

1. *Perivascular sheath.*

The first departure from the healthy condition is usually seen here. Nuclei that become deeply stained in the colouring media are found regularly distributed in the connective tissue. At particular points, by preference in the retiring angles where vessels divide they are found in small groups; but along the vessels

generally they appear to be arranged with tolerable uniformity. Frequently there is seen a disposition for the nuclei to be arranged in a distinct double row on each side of the vessel, one row lying close to the muscular coat, the other near the outer limit of the perivascular sheath. This suggests that the origin of the nuclei is either in the fixed connective tissue supporting the endothelium or in the endothelium itself, or in both. By careful teasing, and the use of high powers, I have satisfied myself that the nuclei are both of connective tissue and of endothelium. Free lymph cells, though abundant enough in the perivascular tissue as the disease advances, are usually absent in the very beginning. The aggregation of nuclei goes on at particular points to form miliary tubercles visible to the naked eye, the general nuclear growth along the vessel undergoing little change.

2. *Vascular lining.*

Soon after the appearance of changes in the perivascular tissue, the nuclei of the endothelium lining the vessel begin to enlarge and become cloudy. Fat globules are seen in the interior of the nucleus, and the whole cell loses its clear translucent character, and becomes granular and cloudy. Occasionally the nuclei divide, but extensive proliferation, such as has been found by Klein in the pulmonary vessels of guinea pigs, I have not yet seen. It is not unlikely that the disease would prove fatal by general meningitis before the pure tubercular lesion had reached this extent. This cloudiness of the endothelium and swelling and division of its nuclei are found in the very smallest vessels as well as in those of medium size. Occasionally a single nucleus is found swollen to such an extent as to extend half way across the lumen of a minute arteriole. Usually the nuclei attain to a very large size before division takes place. This condition of the vascular endothelium is nearly always found when the tubercles on the vessels are large enough to be visible to the naked eye; but it is usually absent till the period when the nuclear groups have begun to form distinct swellings in the perivascular sheath.

DESCRIPTION OF PLATE II.

Plate II. showing initial pathological changes in the cerebral arterioles in acute miliary tuberculosis.

FIG. 1.—Small cerebral artery from which the perivascular sheath has been accidentally removed.

(*a*) Enlarged and deeply stained nuclei of muscular fibres.

(*b*) Nuclei of endothelium swollen, and containing oil globules and granular material.

(*c*) Two nuclei which have undergone division.

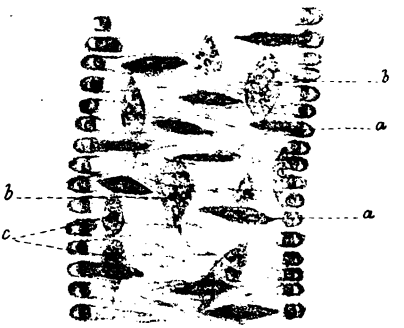
FIG. 2.—Minute cerebral arteriole, showing enlargement of nuclei of muscular fibres and some swelling of endothelial nuclei.

FIG. 3.—Cerebral arteriole and perivascular sheath, the sheath extending beyond the vessel.

(*a*) Nuclei in perivascular tissue becoming crowded at a′ a′.

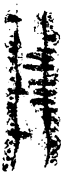
The endothelial lining is somewhat cloudy and its nuclei slightly swollen and granular.

Fig. 1.



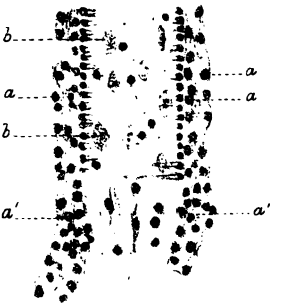
X 1090

Fig. 2.



X 200

Fig 3.



X 200

Drawn by Dr J. Greig Smith.

3. *Changes in the muscular tissue* occur *pari passu* with those in the endothelium. They consist in well marked enlargement, and occasional division of the nuclei of the muscular fibres in the vessel wall. This takes place at irregular points in the length of the vessel, and with no apparent connection with the tubercle groups in the perivascular sheath. Sometimes the swelling of muscular nuclei is so great as to cause in the smallest arterioles a general fusiform enlargement; and very frequently nuclei may be seen with diameters three or four times as large as healthy muscular fibres in their neighbourhood. The large size to which they sometimes attain without division is often very striking.

It will thus be seen that beyond the growth of miliary nodules there is nothing peculiar in the lesion affecting the cerebral vascular tissue. It is simply an inflammation of all the structures of the vessel proper, following on the primary lesion in the lymphatic paths surrounding them. It might be a question whether the marks of irritation in the inner coat arose from poisonous products inside the vessel, or simply by participation through continuity of tissue from the perivascular sheath. It is possible both that the infectious products may be picked up and carried along by the vessel, and that after having saturated the perivascular tissue they might soak through the vascular coats and reach the interior. Be this as it may, the peculiar and specific product of the infection is the miliary nodule in the perivascular sheath; the rest of the lesion is an ordinary inflammation of the structures in the vicinity.

October, 1877.

Contributions to the Pathology of Tetanus.

EDWARD LONG FOX, M.D., F.R.C.P.

DR. Ringer and Mr. Murrell, whilst regarding tetanus in all forms simply as an indication of the diminution in the resistive forces of the cord, point out that some tetanising agents, such as strychnine, leave the reflex action unimpaired, whilst others depress both the resistive and the reflex power. Some agents, for example gelseminum depress the reflex function more than the resistive power, causing considerable paralysis, but slight tetanus. Others, such as box, depress the resistive power early and markedly, and subsequently and in a minor degree the reflex function, the result being strong tetanus with slight paralysis. Strychnine excites powerful tetanus and in large doses depresses simultaneously the motor nerves. Let us therefore accept the term "diminution of the resistive power of the cord," as the physiological condition of the cord in tetanus.

What then are the pathological results found in tetanus? And do they in any way explain the tetanic phenomena? We have first, the dictum of Dr. Wilks and Dr. Moxon, that there are no certain lesions in tetanus.

1.—There is an observation by Mr. Kesteven on a case of idiopathic tetanus, which may bear somewhat on Dr. Ferrier's experiments, and at any rate should lead us to look at other portions of the nervous centres besides the cord. He found the main lesions in the brain. The cortical nerve-cells here were undergoing degeneration and atrophy. There was miliary degeneration of the white matter, immediately subjacent to the cortical grey matter of the convolutions. In the brain, also, there was considerable deposit on the walls of the vessels. In the cord there was nothing notable except thickening of the vascular walls.

2.—Lesions of the peripheral ends of the nerves are met with in a certain proportion of cases of traumatic tetanus; such lesions being absent in many cases, and of course not present in instances of idiopathic tetanus. The nerve too, is often inflamed without tetanus.

3.—There are many and various lesions of the spinal cord frequently met with, involving both grey and white matter, but especially the commissures of the cord and the immediate neighbourhood of the blood vessels.

a.—Inflammation of the neurilemma, traced from the nerves near the wound to the cord.

b.—Granular degeneration of the cells of the cord—stated by Lockhart Clarke to be a constant lesion, but certainly not so.

c.—Softening of spots of the cord.

d.—Hœmorrhage of the spinal meninges.

e.—Bony plates on the spinal meninges. These, however, occur in 50 per cent. of all people that die of any disease above the age of 25.

f.—Effusions of serum into the spinal membranes.

g.—Lockhart Clarke has found irregular areas of disintegration in different parts of the grey substance, and particularly round the central canal, on the right side of which, in one case, was a space of considerable size containing finely granular fluid with debris of blood vessels and nerves. The same was seen in the posterior and lateral white columns.

h.—Dickenson found distension and dilatation of vessels, the vessels crammed with blood corpuscles, escape of their contents, with consequent increase of bulk of cord, laceration and displacement. Clifford Allbutt found the same, with also disintegration of nerve fibres.

i.—Lesions of the sympathetic ganglia have sometimes been met with. In tetanus the vasomotor nerves seem to be

peculiarly sensitive to external impression. Is this due to lesions of a segment of cord, with which the vasomotor nerves are associated, or to some alteration of the blood itself?

j.—I have found in tetanus,—

i. Areas of cord softened, so as to be almost diffluent.

ii. New material beneath the dura mater spinalis.

iii. Hœmorrhage outside the dura mater spinalis, and gummy-looking fluid beneath tarachnoid. Softening of cord and many amyloid bodies in the grey matter.

iv. Distension of vessels and thickening of membranes.

v. Fissures in posterior white columns.

vi. Creamy posterior columns, the softening seeming to be composed of colloid bodies.

vii. Both colloid and anyloid bodies in another case.

viii. Colloid bodies in a case of Mr. Board, fatal in 36 hours from the commencement of the tetanus, 9 days after the accident.

It is probable that all these lesions are the effect of the tetanic phenomena. And not only may tetanus exist without any of these spinal cord lesions, but the abnormalities of the cord may be present without tetanus.

My own belief is that none of the lesions yet found can be considered as in any way causes of tetanus. It is more likely that the blood itself is in fault. In strychnine poisoning, a condition so analogous to tetanus, Dr. Harley has shewn that the blood is incapable of absorbing oxygen in the usual proportion. The immediately fatal phenomena of many cases of tetanus seem to point to some such explanation. This abnormal blood imperfectly nourishes the cord, and either thus, or by its distinctly toxic effects, diminishes the resistive power of that organ. In

other words a cord so conditioned is abnormally impressible. This impressibility renders arterial spasm morbidly facile, whether the exciting cause is the circulation in the cord of more of the morbid blood, or reflected irritation from a diseased nerve at the periphery, or reflex irritation from any other cause and from any other point in the body; and if this arterial contraction goes on for any protracted period, or is frequently repeated, we may find various lesions due to imperfect blood-supply, in addition to those due to diminished nutrition from the original nature of the blood, whilst as a sequence of the spasmodic arterial contraction, we get dilatation, hyperæmia, and perhaps exudation; and lastly the pressure of the exudation, or some peculiarity in its nature, may lead to further disintegration of the nervous centre.

One word, however, as to colloid degeneration, formed in a considerable number of cases of tetanus. It seems to me unfortunate that Sir Wm. Gull and Dr. Sutton use this term to designate mere masses of albuminoid exudations. A comparison of their plates with the one now exhibited, will show manifest differences, apart from the fact that their colloid masses readily take the carmine dye, and these do not. Their description is this "these so-called colloid bodies are probably but simple exudations of albuminoid material (the water of which has been removed in course of preparing the sections). To show that these homogeneous collections, hitherto called colloid masses, are sero-albuminoid exudations, we may state we have observed, when the brain substance has been contused by accident, as in fractured skull, that similar masses are scattered in very large quantity, and similarly diffused into and amongst the nerve elements. In fracture of the spine and injury of the cord, we have found these homogeneous masses in very great quantity in the cord substance. Further, in cases of acute paraplegia (myelitis) a large quantity of this homogeneous material is found lying around the vessels, and disseminated from them into the surrounding structures, with increased nuclei and leucocytes. This condition is simple œdema of the cord. We

have seen similar exudations, but in less degree, in the cord of a person who died of tetanus. In giving this account of these bodies we ought to state that somewhat similar colloid masses may, as is supposed, be produced by dissolution of nerve tubules."

We can readily understand the presence of this albuminoid material in tetanus and in many other diseases. But colloid degeneration of neuroglia nuclei differs widely in appearance.

Dr. Batty Tuke, who holds that they are degenerated nuclei of the neuroglia, states that they appear first in the white matter immediately contiguous to the cortical substance, but as the disease advances they become diffused outwards and inwards. In extreme cases the appearance of sections containing them may best be compared to a slice of sago pudding, for they exist in such large numbers as almost completely to fill the field of the microscope. They are not associated with proliferation of nuclei, and are not colorable by carmine.

Drs. Tuke and McKendrick have experimented on the brains of birds by injuring them with knives and allowing time to heal. In all cases after the 10th day, colloid bodies were found, and they increased in number up to the 19th day. In one specimen shewn, colloid degeneration was found seven days after the accident, and only 36 hours after tetanus had set in.

It is probable that it is a result of inflammatory action, or of hyperæmia just short of inflammation, and as such is also a result of the tetanic spasm, rather than a cause.

February, 1878.

REPORTS.

Dentigerous cyst in lower jaw, from non-erupted wisdom tooth—excision of right half of jaw—recovery.

NELSON C. DOBSON, F.R.C.S.

LOUISA NORFORD, aged 38, a machinist, with good family history, was admitted into the Bristol General Hospital under my care March 31st, 1874. She states that she was always pretty well up to ten years ago, when she noticed a small swelling near her right ear, which caused great difficulty in opening the mouth. About this time she had a double tooth removed (second molar). She does not remember to have had more than one double tooth on this side removed, and can give no history of her wisdom tooth. Her first molar is still present and sound. The tooth also which was extracted was sound, and at the time of its extraction a quantity of "discharge" was let out. The swelling has gradually increased in size from that time until now, but more rapidly in the last few months. The swelling does not prevent her from working,—sometimes it causes difficulty in eating, but never much pain.

At present the patient is in fair health. There is a large oval-shaped uniform hard swelling on the right side of the face, occupying the position of the ramus and body of the jaw, perfectly immovable except with the jaw, and evidently an expansion of the plates of the jaw, as a similar though less degree of swelling can be detected by the finger on the inner surface of the jaw; at one spot on the facial aspect, opposite the first molar tooth, there is a small swelling about the size of a walnut, distinct from the uniform enlargement; and at this place there is fluctuation and some

crackling, as of a thin bony cyst, which is more apparent when one finger is placed on the outer side of the cyst and the other over the alveolar process at the vacant socket of the second molar; the swelling in other parts is firm and resisting. The mucous membrane of the mouth is everywhere perfectly healthy. She cannot open her mouth very wide, but sufficiently so to take food fairly. This being the state of things, and being anxious to try some milder measures before proceeding to excision, I, on April 2nd, after a consultation with my colleagues, made an incision in the mouth over the alveolar process, corresponding to the last two molar teeth. Some glairy fluid escaped; I then passed a long probe through this opening, which made its appearance at the posterior surface of the angle, having passed between the external and internal plates of the jaw. An incision was then made through the skin, and a drainage tube made to follow the probe and kept in position, one end being in the mouth, and the other draining through the skin incision. The bony walls were too thick and unyielding to allow of any crushing. A poultice was applied, and there was a free discharge until May 2nd, when there being no diminution in the swelling the draining tube was withdrawn, and the patient sent out of the Hospital none the better for her treatment.

I kept her under observation, and she was again admitted in September of the same year, and as the tumour was evidently increasing in size and causing her more pain and inconvenience, I determined to excise the right half of the lower jaw. This I did under chloroform by an incision extending from the lobule of the ear along the posterior border of the ramus and under the horizontal portion of the jaw as far as the first bicuspid tooth, and then made a vertical incision from the angle of the mouth joining this; reflected the skin; sawed through the bone at the socket of the second bicuspid which I had previously extracted; I disarticulated at the condyle in the usual way, experiencing difficulty only from the fact that the coronoid process was so enlarged that in

DESCRIPTION OF PLATE III.

Plate III. To illustrate Mr. Dobson's paper on Dentigerous Cyst in lower jaw.

FIG. 1.—The internal portion of the section through the part removed, shewing the central septum and the wisdom tooth at the bottom of the cavity.

FIG. 2.—The outer half of the section, shewing the hollowing out of the external plate of the jaw.

Drawn by G. M. Smith. Natural size.

Fig I.

Fig. 2.

depressing it to get at the insertion of the temporal muscle, it came in contact with the posterior surface of the malar process of the superior maxillary bone; however, I soon overcame this difficulty and there was very little bleeding. The patient's future history was one of uninterrupted progress towards recovery.

Description of parts removed. The accompanying drawing shows pretty faithfully the portion of jaw removed as seen in section.

It will be seen that the portion of the right inferior maxilla behind the last bicuspid tooth was entirely removed.

There was a prominent thin walled cyst occupying the whole of the horizontal portion removed, this cyst being chiefly developed in the outer wall of the body of the jaw. Behind this the whole of the angle and ramus is seen to be uniformly enlarged, the enlargement being most prominent in the facial aspect though it extends also to the oral surface. The coronoid process instead of having its normal somewhat pointed character, is broad, blunt, and expanded, so much so, that it filled nearly completely the Zygomatic fossa, and it was wedged tightly into the anterior part of this fossa.

Turning now to the interior as shown in the internal section, it will be seen that the external plate of the jaw is the most expanded, and the ramus hollowed out in a large cavity which in the recent state was lined by a smooth membrane, resembling mucous membrane, whilst the posterior part of the cyst was cut off from the anterior by a central septum; the walls of this cavity were thickened and especially thick at the upper and lower surfaces. The internal plate shows, though to a smaller extent, structures corresponding to these observed in the outer, but with the important addition of what appears to have been the origin of all the patient's troubles, viz., the fully developed, but non-erupted wisdom tooth, the whole of the swelling and bony expansion being the ordinary dentigerous cyst, unusual only in the fact of its size, and the great bony thickening. The crown of the wisdom tooth was covered by

a dense fibrous membrane, and the cyst's bony walls were so thick that I believe even with the full light of an accurate diagnosis that any less formidable operation than that which I undertook would have been useless. At any rate the attempt to crush the cyst walls and the drainage tube, which with the removal of the offending tooth are generally sufficient in ordinary cases for the successful treatment of dentigerous cysts, gave no relief. There was a difficulty in ascertaining whether one or two teeth had been extracted, but the offending wisdom tooth could not be discovered, though the possibility of its non-eruption was not lost sight of.

Another point worth recording is that my colleague Mr. Parsons with much ingenuity fitted the patient with an efficient substitute for her lost jaw, so that she masticates on that side with comparative ease, the only drawback being that her muscles contract so strongly that the apparatus is constantly requiring repair. This artificial substitute was more readily adapted in consequence of the fact that the genial tubercles of both sides with their attached muscles were undisturbed.

October, 1874.

Cysticercus Cellulosæ found in the lateral ventricle of the Brain.

R. Shingleton Smith, M.D., B.Sc., M.R.C.P.

THIS specimen was taken from the brain of a servant girl, sixteen years of age, admitted to the Bristol Royal Infirmary, on July 28th, 1874. She had complained of feeling unwell for a month, but had continued work till a week before her admission. The first symptom noticed was headache, which came on without apparent cause, and since its first onset had frequently recurred, the patient not having been free from it for a single day. Her general aspect was healthy, but she seemed to be listless and apathetic. Whilst under observation, she complained of frequent attacks of severe pain across the forehead, each attack lasting only a few minutes, with intervals of complete freedom from pain of

DESCRIPTION OF PLATE IV.

Plate IV. To illustrate Dr. Shingleton Smith's paper on Cysticercus Cellulosæ.

FIG. 1.—Dr. Smith's specimen from the lateral ventricle of the brain.

(*a*) The head enveloped in a sac, but indistinctly shewing the presence of suckers and corona.

(*b*) The abdominal sac with an opening in it.

FIG. 2.—Dr. Brittan's specimen from the fourth ventricle of the brain, shewing the thin neck and suckers with corona.

Twice the natural size.

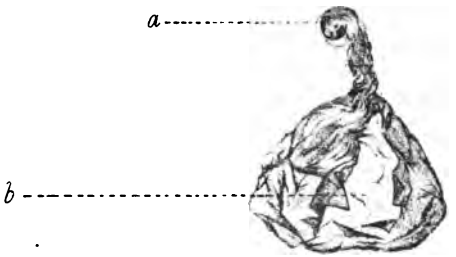


Fig. 1.

Fig 2.

Drawn by George M. Smith.

not more than half an hour's duration. At the onset of the attack there was marked flushing of the face, then copious perspiration, next a fit of crying, and afterwards the attack terminated in a short sleep. She had never bitten her tongue in such an attack, and there was no history of convulsions previously. Vomiting had several times followed an attack of headache. Her sleep was disturbed by the attacks, which usually woke her up several times in the course of a night. There was no complaint of any defect of vision: the pupils were equal and sensitive to light: there was no indication of paralysis. The pulse was 76, the temperature normal.

A fortnight later she became very drowsy: the attacks of headache were frequent, and vomiting took place occasionally. The pulse was 68' regular. The optic discs were examined, and were found to be normal.

On the day following the drowsiness had gone off again, she became much more intelligent, and more free from pain.

On September 3rd the pain had come on again, and vomiting was frequent. The patient was very drowsy, and very apathetic,—feet cold,—pulse 44, small, but regular.

On September 7th the drowsiness had increased, but patient could be roused. The pupils were noticed to be dilated, but equal.

On September 11th the drowsiness was less marked. The pupils were not dilated, and were sensitive,—pulse 48, very small,—extremities cold.

On September 17th, an attack of convulsions affecting the right side of the body was first noticed,—after the attack patient continued drowsy and pupils were dilated,—pulse 68,—the vomiting was more frequent.

On September 20th the patient died in a second attack of convulsions.

At the post-mortem examination, the cyst exhibited was found in the lateral ventricle, loosely attached to the choroid plexus—no other abnormality was observed. The growth consisted of an abdominal sac, three quarters of an inch in diameter, with an

elongated projection from one extremity about half an inch in length. A thin layer of membrane enveloped the whole length of the head and neck, and obscured the suckers and hooklets; but through the enveloping folds, a dark spot could be seen at the distal extremity of the narrowed part of the object, which with a pocket lens resolved itself into a dark central mass with four smaller ones arranged around it. The abdominal sac was distended with a clear serous fluid, but the cervical portion contained an opaque fluid, turbid with granules and oil globules.

In commenting on the symptoms present, Dr. Shingleton Smith alluded to another case in which severe headache and fits of screaming in a girl ten years of age were followed by amaurosis and death from convulsions: after death, a hydatid cyst was found to occupy the anterior lobe of the brain on the right side. The symptoms in both cases were of a very indefinite character, and in both death occurred suddenly and rather unexpectedly.

In connection with this case, Dr. Brittan shewed a specimen which he had obtained from a similar case some years ago, in which the ringed neck, the four suckers, and circles of hooks were all distinctly seen. The following is an outline of the case—

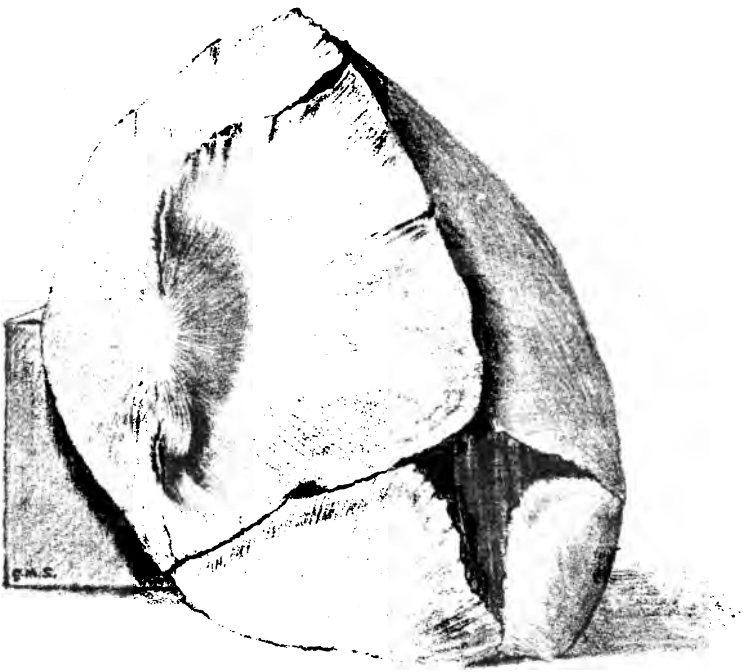
A. M., aged 29, a stout well-made woman, unmarried, having enjoyed excellent health, a year and half ago began to complain of extreme irritation of stomach with vomesis, attacks of severe pain in the head, and febrile excitement,—these symptoms became more intense, and, whilst supposed to be sleeping, she was found to be dead. Her mother had died of some head affection, and a brother also, he having been partially paralysed and unconscious for a week before his death. The cyst, about an inch in diameter, was found in the fourth ventricle, the roof of which was softened and pultaceous—it contained a white semi-opaque mass about one-third of an inch in diameter, from which extended a twisted tube containing fluid, on opening this what appeared to be the head and neck of a tænia protruded.

October 1874.

DESCRIPTION OF PLATE V.

Plate V. Drawing of the skull exhibited by Mr. Leonard, shewing a deep depression in the right parietal bone.

Natural size.



Drawn by G. M. Smith.

Deep depression in the right Parietal Bone of a child born at full time.

C. LEONARD, F.R.C.S.

THE patient had three children previously. The child was still-born after a rather tedious but unassisted labour. There is no fracture, but three or four small fissures exist, produced by separation of the radiating fibres of the bone. There was considerable effusion of blood on the external surface of the skull. The specimen has important medico-legal bearings, it being certain that no external violence produced the depression, as it was present at the birth and for some hours previously. It is remarkable that such an extensive and considerable depression should have resulted from pressure on the promontory of the sacrum before birth.

October, 1874.

Hypertrophy of Thyroid Gland, and successful removal of right lobe.

C. H. DOWSON.

Ada Perdue, aged 13, was admitted into the Bristol Royal Infirmary, on July 8th, suffering from pressure on the trachea from an enlarged thyroid gland. The tumour was not symmetrical; the right lobe being the larger, it bulged on each side beyond the angle of the lower jaw, and a portion of the right lobe overlapped the trachea, feeling like a separate lobule. The right lobe measured 4 inches by 3, and the left was about $\frac{2}{3}$ of that size. Hypodermic injections of iodine, leeches, ether inhalations, etc., were used, but the tumour rapidly increased, and at the same time the symptoms of suffocation became more urgent. On July 28th she had passed a bad night, being occasionally relieved by ether

inhalations; at 1.30 p.m. she appeared pre-moribund with stridulous breathing, and livid countenance, and the immediate removal of the gland was decided upon. Ether was administered, but, as soon as the skin incision extending from the symphysis of jaw to the sternal notch had been made, it was deemed advisable to discontinue the anæsthetic. The parts loosely adhering to the tumour were then separated with the handle of the scalpel, and all firm adhesions ligatured, amongst them were the superior and inferior thyroid vessels. They could not be distinctly felt, but the effect upon the tumour showed that they had been secured. The gland was very adherent to the trachea, and its removal in this locality required some careful and tedious dissection. Immediately upon the removal of the right lobe the breathing was much relieved and the patient expressed herself as feeling better. The operation lasted an hour and a half, much time being wasted in ligaturing every adhesion owing to fear of hæmorrhage from the vascularity of the tumour, the result was, however, that no blood was lost. Considering the relief afforded the patient, it was deemed unadvisable to remove the left and smaller lobe. The part removed weighed eight ounces and a half, the connection between the two lobes was not as large as a crow quill; this was ligatured and divided, it contained a small vessel. The edges of the wound were brought together by horse-hair sutures, and glycerine and carbolic acid dressing was applied. After removal to the ward the patient never showed any troublesome symptoms, she experienced for two or three days some difficulty in swallowing solid food, but after this was able to take ordinary diet. On Aug. 2nd, the left lobe was much softer and smaller, on the 3rd the ligatures came away, and the sutures were removed, on the 12th only a little fulness was left on the left side. and the wound was almost healed. The patient was discharged cured on Aug. 17th, twenty days after operation.

October, 1874.

Lithotomy twice performed on the same subject.

NELSON C. DOBSON, F.R.C.S.

ALBERT MARTIN, aged 2½, was admitted into the General Hospital under my care in July, 1872. He was an exceedingly small, thin and wretched-looking child. His mother states that he has suffered pain in passing water ever since his birth; and on enquiry I found he had the usual symptoms of stone in the bladder of an aggravated character all his life. I sounded him, and discovered a stone which was thought not to be very large. I performed the usual lateral operation, and removed a lithic acid calculus weighing 84 grains. There was no difficulty in the operation. I explored the whole of the interior of the bladder with my finger, and there was no second stone. The calculus was round and somewhat rough. He made a speedy recovery, and left the Hospital quite well of his bladder symptoms, and with his general health much improved.

February 4th, 1875.—Nearly three years after the above operation he again presented himself with all the symptoms of stone. He was now just over 5 years old. On examination a stone was found. This I removed by lateral operation, carrying the incision through the cicatrix of the former operation. The stone proved to be a large smooth lithic acid one, and weighed 328 grains. There was no special difficulty in the operation; and the boy again speedily recovered. During the intervals between the two stone operations he had been twice under my care: on one occasion I removed his index finger, and on the second his thumb of the opposite hand for strumous disease.

The interesting points attaching to this patient's case are,—

1st—The fact that it was necessary to cut him twice for stone before he had reached the age of 6 years.

2ndly—The absolute knowledge that he had no second stone in his bladder at the time of the first operation, thus showing clearly

how rapidly a stone may increase in size in a patient with such a marked constitutional diathesis as this one had.

3rdly—The size of the second stone, which is larger than any I find recorded in the Norfolk and Norwich tables as having been removed by a second operation at such an early age.

I may remark that he was well fed and attended to at home, so that his diet was not at fault so far as I could discover.

February, 1875.

N.B.—Since this case was brought before the notice of the Society, the boy has again been under my observation (in the early part of 1878.) He has grown into a plump respectable-sized lad, but was suffering from incontinence and some symptoms of stone, but no stone could be found, and he improved under medical treatment.

Tumour in the Cerebellum.

CROSBY LEONARD, F.R C.S.

PARTICULARS of this case were given, and the growth was exhibited. The patient was 27 years of age, and the symptoms had been of three months duration. The most prominent clinical feature was the occurrence of intermittent attacks of severe pain in the head, which were occasionally followed by vomiting. There was total absence of pyrexia, and of all indications of paralysis till the last few weeks of life, when double vision was complained of. Two or three convulsive attacks occured, and in the last of these she died.

At the post-mortem examination, the right side of the cerebellum was found to be occupied by a morbid growth, pale in colour, ill-defined, not definitely marked off from the proper brain-substance, soft in consistence, and appearing to be an infiltration of the cerebellar substance. There was no evidence of disease in any

DESCRIPTION OF PLATE VI.

Plate VI. illustrates Mr. Leonard's paper on Tumour of the Cerebellum, shewing the appearance of the cells and of the connective tissue stroma.

x 400.

Drawn by R. Shingleton Smith.

other part of the body. No family history of cancerous disease could be elicited. Mr. Leonard believed that the growth should be looked upon as a glioma.

Dr. Brittan commented on the clinical aspects of the case, more particularly on the difficulty in diagnosis. He considered the disease to be encephaloid cancer; he could distinctly trace wasted nerve tubules amongst the groups of cells of which the growth consisted.

Dr. S. Martyn (to whom the tumour was referred for examination) reported that he found the growth to consist of round cells imbedded in a finely fibrillated stroma, which stroma, under high powers, presented a granular nucleated appearance, and contained small poly-nucleated cell-like bodies readily stained by carmine. He considered the growth to be a glioma.

Dr. W. Moxon subsequently wrote, "I should call the growth a true carcinoma, in fact a typical example. The network is almost as well defined as that of lung tissue. The epithelioid cells filling the interspaces are various in figure, and have large bold nuclei and nucleoli, just as in actively growing specimens."

Dr. Kesteven concurred in the opinion expressed by Dr. Moxon, but he added "that the structure microscopically is very like that of glioma."

March, 1875.

Case of Paracentesis Pericardii.

G. F. BURDER, M.D.

DR. BURDER reported a case of *hydrops pericardii* occuring in the course of chronic heart disease with general dropsy, in which forty-two ounces of serous fluid was removed from the pericardium by the pneumatic aspirator.

The patient—a man aged 60—had been admitted to the Bristol General Hospital, and, the symptoms being extremely urgent, the

operation was performed at once. The relief was very marked and immediate. In introducing the trocar it was noticed that the pericardium (apparently) yielded before it, so that the instrument had to be pushed to an unexpected depth before the appearance of fluid confirmed the diagnosis.

April 28, 1875.

Dr. Burder reported the progress of the case of hydrops pericardii, in which paracentesis was performed.

The patient after remaining in the Hospital ten weeks left in greatly improved health, no fresh effusion having occurred in the pericardium, and the general dropsy having entirely subsided. Cardiac symptoms in a mitigated form continued. He was now in the Hospital again with some increase of the symptoms, but with no indication of a return of fluid in the pericardium.

November 24th, 1875.

Case of Tumour of Cerebellum.

HENRY E. HETLING.

ELIZABETH GRIFFIN, aged 22, single, of dark strumous type, came under observation in July, 1875. Her previous history (a matter of mere hearsay) is as follows:—

Her mother died of phthisis. About Christmas, 1874, she complained of vomiting, which was followed almost immediately by agonizing pains in the head, and by constipation. Previously to this vomiting she had made no complaint of anything, and had followed her occupation. Shortly afterwards she was noticed to stagger in her walk, and had dimness of sight, lost flesh and strength, and menstruation ceased,—all within about a month from her first complaint. Disturbances of vision now commenced. She said the people in the street *seemed to be walking on the tops of the houses*, which I suppose meant double vision. About six

months after this she first came under observation. Her sight was then very deficient, and in about three weeks she had complete amaurosis. She had intense pain in the head, but no vomiting; there was occasional strabismus, and slight convulsive seizures.

The most curious thing was the position in which she was always found, viz., the head was tightly flexed on the chest, the knees flexed on abdomen, and she rested on the vertex of the head and on the elbows and knees; this position she maintained until within a week of her death.

The treatment consisted of Pot. Iod. in gr. x doses, and small doses of Morphia. The latter seemed to do more good than the Iodide.

At the post mortem, the ventricles were found to be distended with fluid, and a tumour occupied the cerebellum. The structure of the entire cerebellum appeared to be destroyed. The surface was nodulated and hard, and the section was creamy, with yellow-grey nodules interspersed.

August, 1875.

Acute Tuberculosis.

E. Markham Skerritt, M.D., M.R.C.P.

THIS case was of interest chiefly as illustrating the relation that might exist between physical signs and anatomical lesions in this disease.

This patient was a woman, aged 27, admitted into the Bristol General Hospital on October 6th, 1875. Her family history was unimportant; she stated that when 10 years old she suffered from "inflammation of the lungs" after scarlet fever. Between the menstrual periods she used to vomit blood. Present illness began seven weeks before admission, with pain in the chest, and shortness of breath, slight cough, no sputa. Since then she had gradually lost flesh and strength, while dyspnœa increased, and her friends told her she was getting very blue. There was occasional diarrhœa,

but no night sweats. On admission, the patient was evidently very weak and short of breath. But the most striking feature was the intense cyanosis present; the surface being of a dusky bluish-purple colour. She complained of some pain below the left breast, increased by cough; cough was very frequent, hard, and dry; there was great tenderness on the site of pain. The respiratory movements were irregular—a number of quick, jerky, very shallow respirations being followed by one or two deep drawn inspirations; movements were exaggerated over the upper part of the chest, but elsewhere diminished. The patient spoke in a half whisper, in a quick, short, jerky way, always smiling; in fact, both breathing and speech were of the "hysterical" type. There was no dullness or percussion. On auscultation there was nothing abnormal except exaggerated breathing over the upper parts of both fronts; there were no *râles* anywhere. The heart's action was very rapid and weak; pulse 160, respirations 50, temperature 102. Next day, coarse dry friction was heard at the site of the pain, over an area of about two inches square. The bowels were opened three times; the motions were loose. There was little change till the 11th, four days later, when cyanosis and dyspnœa were still more marked; no friction was audible; but over the upper part of both lungs anteriorly, as low as the third rib, were heard abundant, very fine sharp subcrepitant *râles*; the breathing was harsh. Over the rest of the chest similar *râles* were more thinly scattered, with harsh breathing. At one spot, on the left front, level with the third rib, bronchial breathing was to be heard. No dulness or percussion could anywhere be detected. The exaggerated movements of the upper part of the chest were replaced by diminished movement, there being little expansion anywhere except at the bases. There was little cough, with scanty sputa. The pulse was 140, respirations 36. From this time up to the death of the patient, four days later, there was no marked change; there was a quiet nocturnal delirium, but no evidence of meningitis. The chief alterations in the physical signs were the following:—at the upper

part of the left front, the percussion note became very slightly higher pitched than the right side; the moist *râles* became rather coarser towards the last, and more abundant over the back. On the last day of life, the pulse was 160, and the respiration 72.

Post-mortem,—The pleuræ were studded with tubercle; patches of emphysema were scattered over both lungs. One section, the left lung, was thickly studded with tubercle; the typical grey granulations being most abundant towards the base, while towards the apex, was chiefly opaque yellowish-white tubercle, some nodules of which were a quarter of an inch across. Patches of lobular pneumonia were scattered throughout the lung. The right lung was in a similar state, except that the granulations were chiefly in the early stage. At the apex was a small cavity about a third of an inch across, full of pus; near it were several cheesy masses. Grey granulations were found in the heart, under the surface of the diaphragm, liver, spleen, and kidneys. In the ileum just above the valve were two small circular ulcers; also in the lowest two Peyers patches, were round ulcers with thickened edges; and in the cœcum two small circular ulcers. The brain was healthy.

It was well known that in acute phthisis the physical signs might vary greatly, both in amount and nature; and in this case there was the minimum of information to be gained by physical examination of the chest. It was remarkable that when the patient was admitted there was no evidence whatever of the existence of any bronchial or pulmonary inflammation. And yet there was sufficient deposit in the lungs to cause a most striking cyanosis, and to result in death a week later. The deposit of tubercle did not necessarily set up inflammation in the tissue around, but in the lungs clinical experience had shown that it was exceptional for no bronchial irritation to be excited. The onset of the lobular pneumonia was shown by increased dyspnœa, and cough, and by the numerous *râles* described; at the same time the temperature became higher.

The course of the temperature (shown in the accompanying chart) did not display the marked diurnal variations often met with in acute phthisis. Before the onset of the lobular pneumonia it had not exceeded 102·6, but then reached 103·4.

It was to be noted that in this case there existed nodules of caseous matter in the lung, which, according to the modern theory of the causation of acute tuberculosis, would be looked on as the centres of infection.

December, 1875.

Cerebral Tumour.

G. F. Burder, M.D.

THE specimen exhibited was taken from the body of a boatman, aged 50. Three months after receiving a blow on the head the patient began to suffer from very severe headache and drowsiness, and then showed symptoms of left hemiplegia. He had no fit at any time. These symptoms increased in severity, and on admission to the General Hospital six months after the accident the patient was hemiplegic on the left side, the face being especially involved, and complained of constant pain in the top of the head on the right side. There was no aphasia nor vomiting, and the mind was clear. He gradually became more and more drowsy, and died on the eighth day after admission, having been comatose for a few hours before death.

At the post-mortem examination the dura mater was found to be adherent to the calvarium over a space of about two square inches a little to the right of the vertex. On removing the dura mater from the brain a firm mass came away with the membrane leaving a well defined cavity in the brain. The brain substance was softened for a short distance around the cavity. No other abnormality was found.

Dr. S. Martyn gave an account of the histological characters of the tumour. It was hard, lobulated, and without a capsule, spurs extended from it into the dura mater in which membrane it originated. It was made up of three elements:—

1. Cytogenous, adenoid, or granulation-tissue, originating in the walls of the vessels occupying the peripheral portion of the tumour.

2. Multipolar cells, myxomatous.

3. Cells and vestiges of fibres in a finely granular (degenerate) basis.

The possibility of the tumour being a glioma, a myxoma, or a sarcoma was discussed.

Dr. Martyn considered that the history was that of a syphiloma; there was strict limitation in size with a tendency to morphological alteration, or rapid transformation of elements; these points tended towards a similar conclusion.

Specimens were exhibited under the microscope of,

1. The intermediate zone of myxomatoid transformation.

2. The granulation-tissue originating in the vascular walls.

3. Vessels cut across shewing the syphilitic new growth around them.

December, 1875.

Rare Placentæ.

A. E. Aust Lawrence, M.D.

THE first placenta exhibited, presented the following peculiarities:

The umbilical cord, instead of passing through the centre of the placenta before it distributed its vessels, was attached to the amnion for eight inches previous to arriving at the margin of the placenta. Whilst running on the amnion in this way, it distributed two large vessels which ran on the amnion, skirting round the

placenta before they finally ended in that structure. A third smaller vessel showed signs of a recent rupture and near an old blood clot, two inches from the edge of the placenta. Another vessel was seen as if it had given way to form the clot. There was a fibrinous deposit under the amnion close to the edge of the placenta, this together with the old blood clot produced sufficient adhesion between the placenta and uterus as to necessitate manual removal. The history of the woman from whom this placenta was removed, was one of great abdominal pain the last three months of pregnancy and two rather severe attacks of hæmorrhage during the third and fourth months. The child was well formed and well nourished.

The second placenta was a very good example of what is known as the "battledore placenta." The cord in this case was attached close to the edge of the placenta, which had a very low attachment to the uterine wall, in fact a portion of it became detached during the labour, constituting a mild form of placenta previa, but happily without any evil results.

The third placenta shown was remarkable on account of the large deposits of fibrous tissue seen scattered over its surface. There was no history of syphilis and the causation of the deposits appeared very doubtful.

April, 1876.

Two Cases of Cerebro-Spinal Meningitis.

H. E. Hetling.

IN bringing these two cases before the Society, nothing, I fear, will be disclosed in either symptoms, course, treatment, or post mortem appearances, that is not already well recognized; and the principal interest centres in the fact that we should have two *sporadic* cases of genuine *epidemic* type occurring in two children, sisters, one sickening but a few days after the other, and no other such cases, so far as we know, in the neighbourhood.

February 19th.—Clara P., aged 7 years, was first seen on the 19th February, 1876. She complained simply of pyrexia; had vomited a little, and said she had a sore throat; the tongue was covered with thick fur; the papillæ were red and prominent; the urine was very slightly albuminous; and it was *said* by the mother that a fortnight before she had had a red rash out. The urine was never again found to be albuminous.

20th.—Screaming with pain in the head; countenance flushes and pales. The head is arched backwards and fixed. There is a spotty petechial rash over the feet and ankles. She had Potas. Brom: gr. x, with Pot. Iod. gr j. three times a day.

25th.—P. 114; T. 101.4.—Had a rigor at 10 a.m. There is excessive hyper-æsthesia. She screams if touched anywhere with the finger.

26th.—P. 114; T. 100.3.—Rigors were frequent. She is desquamating in small branny scales partially over the back, tibiæ and breast.

March 2nd.—P. 120; T. 102.3.—The bowels have been confined for some days, so she had gr. iv. calomel.

3rd.—P. 123; T. 103.1—Passing profuse dark bilious stools, which continued until—

6th—P. 150; T. 105.1.—When two motions passed, pale and yellow. The tongue has been steadily cleaning from *behind* forwards, and is now all clean and moist. Still she seems worse.

7th.—P. 120; T. 101.4.—Diarrhœa returned. Ten stools, olive-green in color. Skin mottled and purple, especially arms and cheeks, with purple veins.

8th.—P. 123; T. 101.3—Bowels moved twice, soft and clay colored. The mother fancies her speech is a little muffled; and so she went on until—

20th.—P. 130; T. 100.3.—When the bowels being confined for many days, she had 3½-oz. doses of hanst. senna co. without effect; and as the belly was swelled and tympanitic, an attempt was made to administer an enema, when it was found that the lower bowel

was completely blocked with solid, dry, putty-like fæces, of which a large quantity was removed with a spoon. An enema was then given, and the bowels acted freely.

23rd.—P. 156; T. 100.1.—She was semi-comatose, difficult to arouse; it was a long time before her tongue was put out after being told. The mother fancies she is a little deaf. A blister to the back of the neck.

24th.—Better; easily roused; pulse better; takes her milk again; speaks frequently. From this time until 31st mentally she seemed to improve. Takes some sherry, and milk in large quantities; but nothing seems to be assimilated. The fæces apparently consist of fine curds of milk nearly unaltered, and she is wasting rapidly. A bed-sore is forming on the sacrum.

April 1st.—She again became drowsy. Blister was repeated, and on—

2nd.—She was again quite conscious, spoke frequently, and understood everything said to her.

4th.—Again screaming with paroxysms of pain, which she has not done for many days. She gradually sank, and died on the—

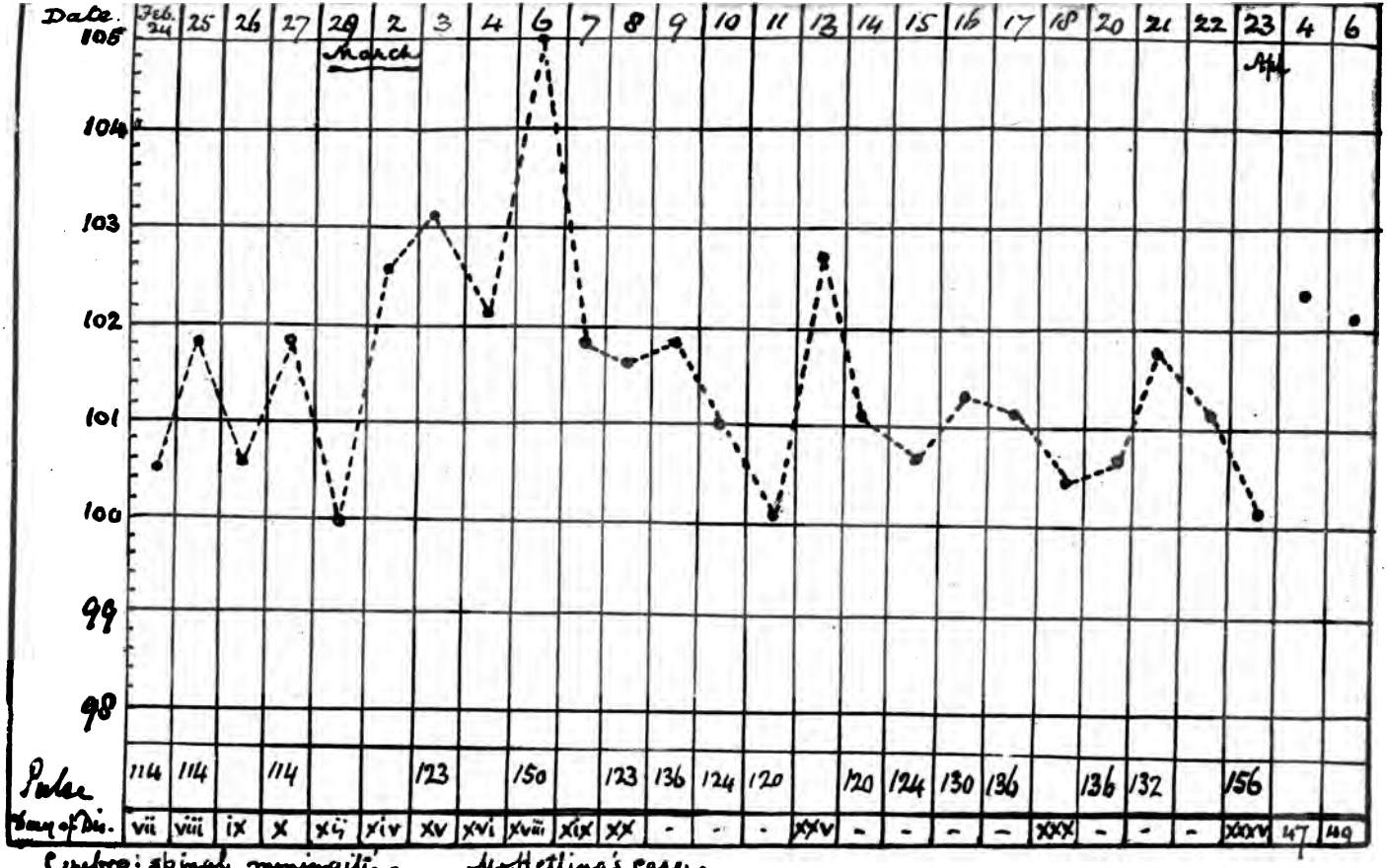
9th.—Wasted to a skeleton, on the fifty-first day from the time she was first seen, and from the commencement of her acute symptoms.

The Case of Lilian P—.

It was on the 19th February that Clara P— was first seen, who then seemed to have had some few days of previous malaise. On the 22nd her sister Lilian, at 5 years, was seized with constant vomiting, furred tongue, and pyrexia. She had a chronic discharge from the ear.

24th.—The head was arched back and fixed; vomiting had ceased; the petechial rash appeared. She also had whooping cough.

April 4th.—She had a spontaneous diarrhœa; dark liquid motions, but occasionally they were quite pale. It will be remem-



Cerebro-spinal meningitis - Mr Hetling's case.

Plate VII

bered that her sister had one or two attacks of diarrhœa, but as she had a dose of calomel I was uncertain how much this had to do with it; but in the younger child it was clear that it formed a part of the natural history of the disease. The whooping cough added so much to the child's troubles that I gave her one grain of quinine four times a day, in hope of checking it. It stopped it completely in four days, when she refused to take any more. (I may add, parenthetically, that this is *not* the first occasion on which I have seen as decided an effect from quinine in whooping cough.)

On or about the 10th May the spasm of the neck began to relax. There had been no complaint of pain for a day or two previous, and gradually she regained her appetite. She is now, I hope, convalescent; but it is ninety-eight days since the commencement of her illness. The recovery continued uninterrupted.

The post mortem examination on Clara P— was kindly made by Mr. H. M. Chute, the House-Surgeon to the Infirmary, on the 10th April, about twenty-four hours after death.

Abdomen discoloured, of a greenish-bluish hue; rigor mortis has passed away; body much emaciated; small bed-sore on the sacrum.

Head. Dura mater, healthy; many of the veins passing from the dura mater to the pia mater along the sides of the Falx cerebri distended with black fluid blood; the pia mater over the whole of the convexity of both sides of the brain intensely inflamed,—even the very smallest sized capillaries were apparent and very much injected. In one spot on the left side in front part of convexity a patch of lymph was evident. The sub-arachnoid space was universally distended with a turbid fluid. The brain on section was healthy, and had *no* increase of puncta vasculosa. The ventricles contained much fluid, the choroid plexus and pia mater of *them* not injected. Ganglia, healthy.

Spinal Cord to middle of dorsal region removed. Dura mater

of upper part pale and natural. About the middle of cervical region, *outside* the *dura mater* and in the cellular tissue between it and the arches of the vertebræ, were numerous small patches of coagulated blood, black in colour; and between the numerous injected capillaries were seen small yellow opaque patches of lymph. The membranes below this point were apparently healthy.

In comparing these cases with those reported by Dr. Cole, of Bath, there are many points of resemblance, but some important ones of difference. They agree in the vomiting, the pain, the constipation, the hyper-æsthesia, the occasional presence of albumen, the spasm, the wasting, and tendency to bed-sore. The differences are, that Dr. Cole makes no mention of petechiæ, of the curious mottling of the skin, and the general venous congestion. In our cases, too, there was no opisthotonos, and it is doubtful if there was any paralysis; but the temperature reached higher than in the Bath cases, viz., to 105.1.

It is curious also to note that on the *twelfth* day of the disease, on the *twenty-third* day, and on the *thirty-fifth* day, the temperature fell to 100°. So far as we could ascertain it did not fall to so low a point on any intermediate day.

It is interesting, too, to observe the great length of time over which the acute symptoms extend. In the convalescing case about seventy days elapsed before the cessation of the pain and the relief of the spasm. One cannot help agreeing with Dr. Cole in doubting very much the influence of drugs; but at the same time counter-irritation to the upper part of the spine was followed on each occasion by at least temporary relief.

May, 1876.

Intestinal Perforation from Ulceration, with Abdominal Tumour of obscure origin.

E. MARKHAM SKERRITT, M.D., M.R.C.P.

THIS case was of interest clinically, owing to the absence of acute symptoms during life; and pathologically, from the obscure nature of a tumour that was found to be present.

The patient was a married woman, aged 47, admitted into the Bristol General Hospital with a history of illness beginning ten days before with chilliness, followed by sharp cutting pain across the epigastric and hypochondriac regions; this pain continued up to admission. Vomiting set in on the second day, and occurred after everything that was taken, the vomited matter being sometimes very offensive. Constipation was complete, and no flatus had been passed. There was a history of three previous similar attacks of constipation, with frequent diarrhœa in the intervals; the first occurred five years before. On admission, the patient had a fairly healthy and well-nourished appearance; the expression of countenance was bright and cheerful, and not indicative of the least anxiety or pain; the pulse was good; the temperature 97°; the tongue was clean; the skin warm and moist; the abdomen was very much distended, and tympanitic; resonance was a little defective over a small area in the hypogastrium. There was entire absence of all pain and tenderness. Nothing further was to be made out by palpation. Soon after admission the patient vomited twice, and the vomited matter was said by the nurse to be brown and unpleasant; after that, however, sickness entirely ceased. The treatment was by laudanum, hot fomentations to the abdomen, and a large simple enema. Next day the general condition was unchanged; an enema of three pints had brought away only a little flatus; the patient said she felt rather better than the day before. Early next morning symptoms of perforation set in, and death followed in seven hours.

Post-mortem examination. The abdominal parietes were adherent to the great omentum, chiefly by old adhesions. The great omentum was spread out over the intestines, and adherent to them, especially below. There was a tumour in the hypogastric region, in the middle line, from its position and symmetrical appearance looking not unlike the fundus of a pregnant uterus at about the fourth month; it was oval in shape, with its long axis downwards, and measured four inches in length, three in width, and about two in depth. The great omentum was adherent to the upper border and sides of this tumour; and from the lower, membranous adhesions passed down over the intestines to be attached behind the pubes. On raising the omentum, fæces were seen in the lower part of the abdominal cavity, and there was intense inflammation of the peritoneal surface of the intestines. Flakes of recent lymph glued the folds of the intestines together; there was a little turbid fluid in the cavity. The intestine was much distended as far as the lower part of the ascending colon, thence to the end it was empty. The peritoneal surface of the cœcum and the contiguous part of the colon was intensely congested and thickly mottled with dark ecchymoses and greyish-black gangrenous patches. A patch at the back of the colon had sloughed out, leaving a hole about three quarters of an inch across, through which fæces freely oozed. On opening the intestine, the mucous membrane of the ileum, chiefly at the lower part, was found to be inflamed, and Peyer's patches were slightly swollen. The mucous membrane of the whole of the cœcum and the adjacent part of the colon was intensely inflamed, and there were gangrenous ulcers on the surface, with sloughy and blackly-ecchymosed bases and edges; one of these ulcers had given way. There was a tract of old ulceration, and the mucous membrane was thickened.

The tumour was found to have a narrow pedicle about an inch and a quarter long passing downwards from its lower aspect; and at first sight this appeared to be connected with the end of the vermiform appendix; the pedicle was twisted on itself so as to

cause strangulation of the tumour. The close adhesions of the body of the tumour would have prevented it from rotating on its own axis, and the strangulation must therefore have been produced by twist of the other end of the pedicle, owing to the movements of the distended intestines, while the tumour itself kept its original position. The appendix was pervious for two inches, and healthy; closely adherent throughout to the pedicle. Just beyond the end of the appendix, there was a small cyst in the pedicle, which might be taken to represent a piece of the appendix cut off from the rest; if so, the hypothesis would suggest itself that a similar dilatation of the extremity might have been concerned in the production of the tumour. However, on dissection, the bulk of the pedicle appeared to be mesentery, to which the attachment of the appendix was accidental. On section the tumour was found to be a cyst, with a thick fibrous capsule, and a smooth lining; the substance of the wall was composed of laminæ of tough fibrous tissue, in parts arranged somewhat concentrically, in others looking as if crumpled up and put inside the capsule. Thickened parts of the wall projected irregularly into the cavity of the cyst. The latter was full of a chocolate-coloured paste studded with glittering points. Microscopically this paste consisted of granular matter, fat globules, and cholesterine crystals. The cyst-wall was composed of fibrous tissue, in some parts well formed, in others almost structureless and finely granular; in the meshes of this tissue were a few small round granular degenerate cells. No characteristic hydatid structure could be found.

The chief clinical interest of this case was in the entire absence of all acute symptoms of abdominal mischief up to the occurrence of perforation. The history and the symptoms led to the conclusion that no acute abdominal affection was present; hernia, internal strangulation, intussusception, acute enteritis from any cause, seemed to be excluded by the total absence of urgent symptoms;—a clean tongue, a good pulse, a cheerful and fairly healthy aspect, the absence of vomiting, and the absence of tender-

ness, appeared to negative the existence of these conditions. There remained the causes of chronic or non-acute obstruction—chiefly stricture and fæcal accumulation. With regard to stricture, gradually produced and suddenly obstructed, the history of frequent diarrhœa was the chief point in its favour; but, on the other hand, there were no strictures to be discovered, no specific history, no cachexia, no peculiarities of the stools, and the history of repeated attacks of constipation extending over five years did not favour this view. The above conditions being thus eliminated, the conclusion was come to, that fæcal accumulation was the probable cause of obstruction; the history was certainly in accordance with this conclusion—several previous attacks of constipation, lasting from seven to ten days, not accompanied by acute symptoms, and entirely passing off after use of enemata.

The *post-mortem* examination showed that acute enteritis alone was the cause of the obstruction, as the inflamed tract of intestine no longer continued its peristaltic action. Ulceration had set up intense enteritis, and this must have been present at the time that there was a total absence of all acute symptoms. It was probable also that considerable peritonitis had existed at the same time, for it was not easy to see how enteritis affecting the whole thickness of the gut could have been present without it; and the lymph that glued the intestines together was so copious and well-formed that it could hardly have been deposited only during the seven hours that intervened between the occurrence of perforation and the death of the patient. It was to be remembered, however, that after operations for hernia, lymph was sometimes found within a few hours. The presence of old adhesions showed that peritonitis must have existed in previous attacks, which according to the patient's account were not accompanied by any marked acute symptoms. It was well known that local symptoms might sometimes be almost absent when acute abdominal mischief existed; but usually then, the general state of the patient would indicate the presence of very serious disease; whereas here, both

the local symptoms and the general condition were the reverse of those characteristic of the affection.

The case also illustrated a point in the treatment of intestinal obstruction; that in all cases it was prudent to avoid giving purgatives by the mouth, inasmuch as the symptoms might not indicate the gravity of the actual condition.

As to the tumour—how and where did it originate? And what was its primary nature? The bulk of the tumour probably originated in a blood-clot, the fibrin of which had become deposited in laminæ, while the rest of the blood remained as the chocolate-coloured cyst-contents. Whether the hæmorrhage took place into a pre-existing growth, or into the meshes of the mesenteric tissue, could not be determined. Rokitansky had described a condition where the vermiform appendix got blocked up and dilated into a kind of dropsical pouch; if that had taken place, and hæmorrhage had occurred into the cyst thus formed, some such structure might have resulted. The greater part of the appendix, however, was pervious and healthy, and, as before stated, the balance of anatomical evidence was against the view that the tumour originated in the appendix.

May, 1876.

Epithelioma of Tongue, with secondary growths in the Lung.

DR. SHINGLETON SMITH.

THE author exhibited these specimens at the meeting on October 25th, 1876, and at the following monthly meeting gave an account of the microscopic appearances.

The specimens were taken from the body of a gentleman aged 72, who had suffered for several years from some disease of the tongue, and who at last died in consequence of a gradually increasing difficulty in swallowing, and weakness partly resulting

from the constant flow of saliva and mucus from the mouth.

As regards the history of the case Dr. Beddoe writes: "The only points likely to be of interest are,—1st, the alternate progress and retrogression of the mass of disease on the superior aspect, the retrogression apparently due to the local action of caustics and astringents. The portion of the growth which used to give him pain by getting between his molar teeth became quite atrophied latterly, while downwards the increase continued. 2nd, as regards pulmonary symptoms. For some months there had been a certain quantity of expectoration, hardly however to be distinguished from regurgitated food, secretion from fauces, &c. For a week or ten days before death he complained occasionally of sharp pain in the right side:—this may have been muscular or pleuritic."

At the post mortem examination the tongue was found to be almost destroyed by a large ulcer,—only the tip had escaped. The base of the ulcer was much indurated, and numerous nodular masses were scattered through it. The ulcer extended back to the epiglottis, and dipped down between the left glosso-epiglottidean fold and the median line. The larynx was healthy, but the epiglottis was much more folded than is normal, forming an elongated V-shaped aperture. Three hard nodular masses were found in the substance of the upper lobe of the right lung, and a caseous patch on the surface of the pleura. Sections through the base of the ulcer, and through the nodules in the lung exhibited a typical epitheliomatous structure. The indurated mass forming the remains of the tip of the tongue consisted of fibro-cellular tissue infiltrated with epithelial nuclei: no muscular fibres remained; no muscular structure was visible. The epithelial layer was much thickened, forming large papillary growths; and in the deeper layers of this epithelial covering could be indistinctly seen the roughened margins of the "prickle-cells," or "conjoined epithelium" as it was named by our late President, Dr. S. Martyn. All the sub-lingual tissues, more particularly the glands, were infiltrated with a small celled epithelial growth.

DESCRIPTION OF PLATE VIII.

Plate VIII. illustrates Dr. Shingleton Smith's paper on Epithelioma of Tongue, with secondary growths in the lung.

FIG. 1.—Rounded nest-like mass from the lung, composed of very large epithelial cells imbricated at the margin.

FIG. 2.—Conjoined epithelial cells from the deeper layers of the epithelial covering of the tongue.

Drawn by R. Shingleton Smith.

Fig. I.

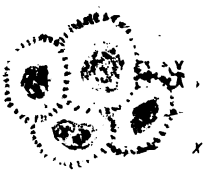


Fig. 2.

The point of principal interest in this case was considered to be the secondary implication of the lung. The author of the paper had seen only two cases of epithelioma of the lung: in both the lung disease was secondary to an ulcerating epithelioma of the tongue. In both epithelial fragments may have been separated, and have given rise to mechanical infection. The case was, therefore, believed to afford another illustration of an unusual mode of infection by a transplantation of epithelial particles from the tongue to the pulmonary tissue, by descent of cancer-germs down the bronchial tubes (Dr. W. Moxon, *Path. Trans.* Vol. XX. 28.)

October, 1876.

Specimen of Ovarian Cyst, together with the uterus from the same person, shewing two large fibroid tumours.

A. E. Aust Lawrence, M.D.

THE cyst was removed from a woman aged 46 years, who had suffered from ovarian disease for two years, and whose symptoms had become so urgent as to necessitate ovariotomy, as giving her the only chance of life. After the operation, which was difficult on account of numerous adhesions, both parietal and visceral, the patient did very well for seven days, after which time she gradually sank and died on the ninth day, apparently from exhaustion, as the post mortem examination did not reveal any more than slight peritonitis.

The uterus was removed, post mortem, and on section showed very well two large intra-mural fibroid tumours.

Dr. Lawrence alluded to the fact that it is not uncommon to find a fibroid uterus as well as ovarian disease in the same patient.

October, 1876.

Two Surgical Cases.

AUGUSTIN PRICHARD, F.R.C.S.

Case I.—*Recurrent fibroid tumour of the breast.*

I WAS consulted early in 1866 by a maiden lady, aged 65, about a tumour of the right breast. It was large, prominent, smooth, soft, and elastic, not adherent to the skin or subjacent parts, and had been growing for two years. Her general health was good, although she was of a spare habit; and there was no history of cancer in the family. I gave her my opinion that it was non-malignant, but advised an operation. She then, as so often happens, went up to London to Sir J. Paget, who told her to come back and submit to the operation, which was done on the 9th of April, 1866.

After making the long necessary cut through the skin—intending to turn the tumour out from its bed—upon grasping it, my fingers sank into its substance which was very soft and black, almost like a broken down coagulum, and very free bleeding ensued, so that the patient became very faint, and I was obliged to complete the operation as quickly as possible, using a great many ligatures to tie the vessels. There was little or no suppuration afterwards and but little action; and during the healing of the wound, I repeatedly washed it out with the chloride of zinc solution we use for such purposes, and the case at first appeared to get well.

The mass removed consisted of a sac containing dark semi-fluid substance, and which was made up chiefly of spindle-shaped cells with but little fibrous structure.

Her general health improved, and she had no pain; but the respite from the malady was brief, for in six months' time the disease had returned to such an extent that I advised and performed another operation, removing a smaller mass and every suspicious portion that I could find. There were two or three isolated nodules of the same dark morbid deposit in the neighbourhood of

the principal mass, which were of course taken away, and the wound was washed out with the chloride of zinc. She bore the chloroform very well and soon made a quick recovery. This was *November*, 1866; and in *May*, 1867, the same thing occurred with the difference that on this occasion the disease was adherent to the fascia over the pectoral muscle, which was necessarily divided and partially removed. I saw my patient in March, 1868, when she was apparently well, after an interval of ten months; but after three months more had elapsed the disease returned and the same routine was carried out, and I operated again for the fourth time in June of that year.

My patient was a placid, courageous old lady, readily submitting to anything that I recommended; she had no dread of the operation itself, and her recovery was generally speedy, on some occasions complete union by the first intention taking place.

The subsequent history of the case was this:—I operated a fifth time in March, 1869; sixth, in November, 1869; seventh, in May, 1870; and eighth, in January, 1871. The morbid growth re-appeared at the end of that year, but it was so adherent to the subjacent parts that I did not advise any further operation, and it grew at first slowly, but afterwards more rapidly, and spots of ulceration formed in its surface, from which she had repeated attacks of bleeding. She died in June, 1873, in her seventy-fourth year. Her general system was to the last unaffected by the disease.

There was no post mortem examination, and indeed none was necessary, for there were no signs of any disease of the internal organs; but the inspection of the body shewed that the tumour had attained an enormous size, extending over the whole of one side of the chest, lobulated, very prominent, ulcerated in parts, and vascular. I consider this remarkable case worthy of notice because it was typical of recurrent local disease, obviously not of a cancerous nature, and not infecting the constitution of my patient: and from a practical point of view I think it not improbable that if the cyst had not ruptured and scattered abroad its contents as seeds for

future growth in my first operation, the disease might have been checked, and the patient cured at once; and the case also shows, as will my next one, the power of old people to bear surgical operations; and although I have a well-founded dread of chloroform, I cannot but believe that both my patients were more tolerant of these frequent operations, because their nervous systems were preserved by this agent from all shock.

Case 2—*Vesical calculus.*

In the beginning of the year 1867 an old gentleman came under my care suffering from symptoms of stone in the bladder. His troubles had been of long standing, and had become very urgent. He lived a considerable distance from Bristol, but was unwilling to leave his home for lodgings either in Clifton or London. He was seventy-five years old, very deaf, had been operated on in London for cataract in both eyes, one of which was afterwards extirpated, and he had met with a severe injury to his left hip and thigh, the exact nature of which I never made out, but which caused anchylosis of the joint on that side,—an important item in the choice of an operation for stone.

The sound on entering the bladder came at once upon a hard and large stone, and it was determined to attempt lithotrity, which was complicated by the condition made by the patient that he should always have chloroform.

My first attempt to crush the stone was in May, 1867, in which month I operated three times with the effect of only taking off some of the outside of it, for when I fairly grasped it by opening the lithotrite to its greatest width, my own strength and the power of the screw were at first insufficient to break it up; and on one occasion I was dismayed by finding that the upper blade of one of Weiss's strong lithotrites had been bent by the force used.

The fourth operation was in June, three more in July, in which month we managed to crack the offender across and thus facilitate our proceedings. I operated twice in August of that year, twice in October, and once in December, making twelve operations.

On each occasion a considerable amount of debris passed along but there were still large and hard knobs to be broken up. The patient was now much relieved of his former pain, and was always willing to undergo his ordeal; but the quantity seemed so large and the stone so hard that once or twice I felt disposed to tell him that he had better let it all alone. This however was not done, and I continued my work in his bladder during the year 1868, operating once in January, once in February, twice in March, once each in April and May, and three times in June, when I found on one occasion some large pieces impacted in the urethra, which were dislodged with some difficulty; once again in July and once in August, making eleven times in that year, or twenty-three times altogether. On the last occasion I did not crush any stone, but my visit was because there was a piece impacted which I could not move by the usual means: I therefore cut into the urethra, and with my finger turned it out, and then I sounded him and found no stone: the last piece had stuck fast, and required a free incision to remove it. When the wound had healed the case was ultimately cured, and had no trouble for four years, when I was called to see him, for he had partial retention of urine. A stricture had formed where I had opened his urethra, and some inflammation and suppuration existed behind it, and a probe only would pass. I stretched it fairly with Barnard Holt's instrument, and he was entirely relieved. He had little or no trouble with his urinary organs afterwards, and died last year, I believe of some bronchial affection, between 80 and 90 years of age, about eight years after the last lithotrity operation. I am not aware of any special practical deductions from this case, except that it shows the advantage of perseverance,—an essential attribute of a good surgeon; and I must add that in this particular I was much helped and encouraged by the surgeon who had the daily charge of the patient, who assisted at all the operations, and in fact bore the brunt of the general treatment, as well as the management of the numerous local troubles arising in the course of such a long and difficult case.

March, 1877.

Notes on a case of retained Menstrual Discharge.

A. E. Aust Lawrence, M.D.

THE subject of this case was a woman, aged 33, married, and the mother of three children, the last born fourteen months ago. On admission to the Bristol General Hospital she complained of difficulty in passing water, and great discomfort when the bowels acted, also pains in the abdomen and thighs like labour pains.

Her previous history, which might throw light on her present state, was that she was confined at a workhouse fourteen months ago, and had an illness for two months after the confinement. She had not menstruated since her confinement. Although married, she did not after this confinement live with her husband.

Upon examining the abdomen after emptying the bladder, there was a firm roundish swelling about the size of one's fist, above the pubes. A vaginal examination revealed numerous bands of cicatricial tissue stretching obliquely from the anterior to the posterior vaginal wall. The vaginal roof was drawn up into a cone, at the apex of which was felt a small portion of hard tissue, about as big as a horse-bean. When pressure was made by the finger upwards in the roof of the vagina it encountered almost everywhere a more or less dense body, which appeared fixed in the pelvis. There was no trace of cervix uteri to be felt, and no opening or depression to indicate the situation of the os uteri, and the only place where anything like uterine tissue could be felt was the small hard nodule already mentioned.

The diagnosis made was menstrual retention, due to closure of the os uteri externum from inflammatory mischief resulting from injuries after confinement.

Within a few days the swelling in the abdomen became very much larger, and the woman complained of very intense pain,—in fact her condition was one of great danger, and it was decided to

make an exploratory puncture into the uterus, per vagina. She was placed on her left side, and a Sims' speculum introduced and guided by the finger; an aspirator needle was passed through the bit of hard tissue in the apex of the vagina upwards and forwards to the extent of two inches, when it passed into the cavity of the uterus, and there was drawn off the characteristic dark treacly menstrual blood. The aspirator needle was now withdrawn, and a trochar and canula passed, through the latter of which the fluid was allowed to drain slowly off to the amount of about twenty-five fluid ounces. When the fluid had ceased flowing, the uterine cavity was washed out with Condy's fluid. The after treatment consisted in daily washing out the uterus with Condy, and keeping the canal open by passing a gum elastic catheter daily. Ergot was given in 20 minim doses twice a day, and the patient kept in bed for two weeks. Her recovery was perfect, and she now menstruates without any difficulty, although she formerly suffered from dysmenorrhœa, the pain at the periods being due, I expect, to a narrow cervix, which the operation has remedied.

March, 1877.

Case of Glioma of Retina.

ARTHUR W. PRICHARD.

TOWARDS the end of July 1876, a child three years old, from Yate, was brought to the Bristol Eye Dispensary by his parents who noticed that the pupil of the right eye never had been as clear as the left, and that it was getting worse.

On looking at the child a yellowish reflection from behind the pupil of a metallic brilliancy was noticed. The iris was sluggish, and the pupil large. With the ophthalmoscope I could see a grey mass obscuring the disc, and extending along the outer wall of the eye; the surface was covered with small prominences and dark

brown spots, and no vessels were visible; the retina to the inner side of the eye was apparently healthy.

In a fortnight's time the disease had increased and the pupil was blocked up with a mass lying apparently close behind the lens. The vessels in the schlerotic were injected, and the outer side of the eyeball was slightly bulging, the prominence giving to the touch a feeling of solidity.

The case being diagnosed to be one of cancer of the eyeball, removal of the globe was determined on, and on August 6th, I extirpated the eyeball, and the child went home four days afterwards quite recovered from the operation.

I did not see the child again till April 18th, 1877, when his father told me that he had been quite well through the winter, until three or four weeks previously, when a growth at the back of the socket of the eye was noticed. This had been rapidly increasing, and when I saw him a hardish white tumour filled the orbit, and was pressing forwards from behind, and prevented the child from quite closing the lids. The vessels on the lids and brow, as well as on the growth itself, were large. The child apparently suffered no pain, and was, as he always had been, in excellent general health and spirits.

I saw him again a week later, and the tumour was larger and more vascular, the surface was tolerably even and hard, and not attached to the lids. There had been no bleeding, but the child complained of pain in the head, and vomiting for three days; he had not lost flesh. He belonged to a healthy family on his father's and mother's side.

On examining the eyeball a mass of soft consistence was found growing from the neighbourhood of the optic disc, and along the outer side as far forward as the ciliary region, and adherent to the posterior capsule of the lens. The vitreous was fluid, and contained portions of the tumour floating in it, which ran away as the globe was opened; the lens and cornea were quite clear, and the other parts of the eye to all appearances healthy.

Dr. Shingleton Smith kindly assisted me in examining the eye and we found the tumour to consist of round nucleated cells, and granular matter. It was very soft before being hardened in spirit, and vessels were present, but in the sections which were from the surface we could recognise scarcely any solid matrix. In the sheath of the optic nerve no cells were to be seen, but there was great difficulty in cutting a section of the nerve itself, as the portion attached to the globe was short and had retracted into the schlerotic during the hardening. The case from its history and appearances is a typical one of round celled sarcoma, or glioma growing from the connective tissue of the optic nerve or retina, and recurring in the neighbourhood after removal.

April, 1877.

Case of Nasal Calculus.

ARTHUR W. PRICHARD.

THIS case occurred in an out-patient at the Infirmary, and I have brought it forward because I believe such cases are very rare.

Susan Tratman, aged 22, came under my care on the 10th of April, 1877, saying she had a polypus in the nose. With a probe I found that a hard mass situated in the inferior meatus filled up the left nostril, and I charged her with having pushed a stone or some other hard substance into her nose. This she denied, but said she fell down some distance on her face at Weston among the rocks eleven years before, and her nose bled considerably at the time. After that she had bleeding from the nose at long intervals; and three years previously at the Infirmary a polypus was removed from the same side. That operation enabled her to breathe comfortably through the nostril, but it soon filled up again, and for almost three years she had suffered from frequent frontal

headache, complete stoppage of that side of her nose, and epiphora. Her nose was very slightly bulging on the affected side, and the patient said it had altered very much in shape, having lately grown much larger and become somewhat "Roman."

I took hold of the mass with a dressing forceps and found it was partly movable, and in endeavouring to extract it, broke off three or four small pieces and pulled them out, but could not take away the whole as the patient was so restless. There was very little bleeding.

A week afterwards she came again, and I extracted the calculus with a fine forceps; the external aperture not being very large rendered extraction somewhat difficult. The operation was followed by very little hæmorrhage, and she could blow freely through both sides. A week later I carefully examined her nose, there was no more stone there, and she was quite well.

On examining the specimen I found a considerable piece of old yellow silk handkerchief firmly attached to the middle of the larger piece and to one of the fragments. The stone is very hard and consists of carbonate of lime and magnesia, coated apparently with phosphate. It measures nearly an inch in length, half an inch across, and one-third of an inch in thickness.

Doubtless the silk was a nucleus round which the concretion formed, yet the patient never remembers having her nose plugged, nor did she ever push a piece of handerchief into her nose.

April, 1877.

A Case of Abdominal Aneurism becoming diffused after Distal Compression.

E. Markham Skerritt, M.D., M.R.C.P.

SPECIMENS were shown from a case of aneurism, of which the following is an account.

The patient was a man aged 31, admitted into the Bristol General Hospital on November 2nd of last year. He was a clerk,

a native of Bristol; had never indulged in violent exercise; there was no history of syphilis. Four years ago he had rheumatic fever; there had been no dyspnœa until lately. The present illness began about seven months before admission with pain over the liver and tenderness; soon after there was epigastric pain, and a stoppage of the bowels for seven days occurred, which yielded to purgatives; the pain was then relieved. Similar pain began to come at times on each side, about the umbilicus, or in the back, gnawing in character, several times shooting down the right thigh. For the last fortnight especially the epigastric pain had been worse after food, coming on almost directly after a meal, and sometimes lasting for hours. There was no nausea or vomiting. The patient said he had lost 2½ stone in weight since the illness began.

On admission the abdomen was flat; the muscles were tense; epigastric pulsation was visible; the aorta could be felt beating rather forcibly from the tip of the ensiform cartilage as far as the bifurcation, but most plainly two inches below the tip of the cartilage; here there was a little tenderness on deep pressure. No abnormal lateral expansion could be defined. A *bruit* was heard along the aorta from half an inch below the ensiform cartilage for three-and-a-half inches, most marked two-and-a-half inches from the tip, when it was rough and rasping; much intensified by the least pressure; inaudible in the back. The iliac arteries pulsated in a similar way to the aorta, and a murmur was produced in them by very light pressure. The heart's impulse was weak; the sounds normal.

A fortnight later the pain had become much more severe, occurring in violent paroxysms, chiefly in the right hip and thigh. A thrill could now be felt in the epigastrium, two inches below the ensiform cartilage, where some expansile enlargement of the aorta could be detected. The *bruit* was much louder, and could be faintly heard in the back, opposite the first lumbar vertebræ. During the next month the pain increased in frequency and violence, and the tumour became larger and more distinct. As

the aneurism was making rapid progress, the author resolved to try to cause coagulation in the sac by compressing the aorta *below* the aneurism, the situation of which made proximal pressure impossible. At the same time it was fully recognised that the prospect of success was not great, and that there would be considerable risk, owing to the increased pressure that would be produced in the sac. Accordingly, on Dec. 13th, the bowels having been freely opened, ether was administered, and at 10.50 a.m. Lister's abdominal tourniquet was adjusted, and the pad was slowly screwed down till pulsation ceased in the femorals; the middle of the pad being an inch and a quarter above the umbilicus, and two inches below the lowest part of the tumour that could be felt. The pulsation in the aneurism at once became much more forcible, and the tourniquet was therefore slackened till the pulse could be felt in the femorals. Pulse 104, regular. In ten minutes the pad was again down so as to stop pulsation in the femorals. At 11.26 the pulse was 108, weak and small; the tumour was rather less distinct, and no thrill could be felt, but the *bruit* was unchanged. At 11.48 the thrill was again felt. At 12.40 no thrill was to be detected. At 1.10 there was a slight intermittent thrill, and the pulsation of the tumour was decidedly less marked. At 2.30 the general condition seemed as before, but on putting the hand on the lower part of the thorax on the right side there was to be felt distinct pulsation, forcibly expansile, extending from the tenth right interspace to two inches above the iliac crest, backwards to the edge of the quadratus, and outwards as far as the posterior axilary line, corresponding to a distinct rounded bulging of the surface. The tourniquet was removed, and the pulsation in the femorals was at once restored. The patient had been kept quietly under ether for four hours without any difficulty; and there were no signs of disturbance from the compression, except that the urine passed soon after contained a trace of albumen. Next day the epigastric pulsation was less distinct, but wider; the tumour in the right loin was beating forcibly over a rather larger area, and a faint *bruit* was heard over it, apparently conducted from the aorta.

During the next three weeks the pain became more frequent and intense; and at the end of this time the local condition was as follows:—on the right side posteriorly there was an egg-shaped pulsating swelling between the ribs and the iliac crest, about three inches long by two wide, visible on the surface, tender. On the left side, in a similar position, a pulsating tumour was now felt, bounded above by the line of the last rib, below by the iliac crest, and reaching outwards for three inches from the spine; tender at its outer part. A *bruit* was heard over the tumour on both sides. If the hands were placed one on each side of the body over these prominences, they were distinctly separated at each beat of the heart.

From this time the patient gradually sank—pain continued—emaciation became extreme—slight œdema appeared over the swelling on the right side—and he died on January 20th, nearly seven weeks after the compression of the aorta and the diffusion of he aneurism.

Post mortem examination. On opening the abdomen, the peritoneum was found to be pushed over to the left as far as a line one inch inside the anterior superior iliac spine by a tumour lying behind it, and the viscera were consequently displaced to the left.

On removal of the stomach and intestines the tumour was exposed. It lay behind the whole abdominal cavity, and beneath the psoas and iliacus muscles. It might be described as composed of two large thick conical masses, with their bases upwards, united at their sides for two-thirds of their length, while their free ends passed downwards to either thigh along the brim of the pelvis. The right mass was the larger, being about four or five inches thick, and reaching up to the liver; its right border was uncovered by peritoneum, and filled the whole space between the abdominal wall in front and the abdominal wall behind. The tumour consisted of a large quantity of soft dark clot and dark fluid blood, shut in simply by the muscular layers of the abdominal walls strengthened by fibrin deposited amongst them. The posterior

wall of the aneurism was formed of the eroded spine in the middle line, and at the sides the muscles and other tissues bounding the body behind. On the left side a conical cavity full of clot passed down beneath Poupart's ligament along the psoas into Scarpa's triangle, and then downwards and backwards for three inches towards the perinæum. The aneurism had sprung from the back of the aorta just below the diaphragm; its mouth was a smooth rounded opening three-quarters of an inch across. The limits of the primary sac could not be made out. The spine was deeply eroded at the last dorsal and first two lumbar vertebræ. The aorta was atheromatous, the arch slightly dilated. The heart was healthy.

The author remarked that there were four chief ways in which aneurism of the abdominal aorta might be treated:—by rest and diet simply; by drugs; by galvano-puncture; and by mechanically more or less preventing the flow of blood through the sac. It was not found practicable here to diet the patient strictly; he always felt much worse when taking iodide of potassium; galvano-puncture was not admissible, as the aneurism had nowhere shown signs of approaching the surface; as, therefore, the patient was growing rapidly worse, there remained no choice but either to let him die or to try the effect of compressing the aorta beyond the aneurism. The distal treatment of aneurism which we were most familiar with was that where a permanent ligature was put round a vessel or vessels beyond the sac. This, as was well known, had been done for aneurism at the root of the neck, where the carotid had been tied for aneurism arising close to its origin; or where the carotid, subclavian, or both vessels, had been ligatured for aneurism of the aorta or the innominate. But if proximal compression had to a great extent taken the place of the old Hunterian operation, there was no apparent reason why compression beyond the aneurism should not be used in suitable cases instead of ligature. Where the aneurism was high up on the abdominal aorta we were limited to temporary compression of the vessel below, with the object of producing rapid coagulation in the tumour.

What chance of success had this distal compression of the abdominal aorta? One of the earliest cases in which this method was tried was Bryant's, where Lister's abdominal tourniquet was applied for twelve hours, removed for twelve hours, and again applied for four; at the end of that time alarming symtoms set in, and the patient died in eleven hours. *Post mortem*, it was found that the intestine had been bruised by the pad, and had set up fatal peritonitis; but that the aneurism, involving the coeliac axis and the superior mesenteric, was consolidated. In the few other cases on record no such result followed; but this one instance was enough to justify the proceeding and to allow hopes of success; although theoretically the chance of consolidation was small; because in such a case the great probability was that free circulation would be kept up in the aneurism owing to the origin of large vessels either from the sac itself or between it and the point of compression. What were the dangers of distal compression? The only one not shared by the proximal operation was the risk of rupture of the aneurism, owiug to the increased pressure on its inner surface caused by the arrest of the flow through the aorta beyond. This accident happened in the case related; and another instance was reported in the British Medical Journal of April 21st, 1877, where the abdominal aorta was being compressed by Heath of Manchester for aneurism of the femoral, when death resulted from rupture of an unrecognised abdominal aneurism above the point of compression. In the author's case, the rupture took place into the retro-peritoneal tissue; and therefore death did not at once follow, but a false aneurism was formed, which attained an enormous size. It was worthy of note however, that in cases of distal ligature at the root of the neck, there had often not been increased pulsation in the sac, but, on the contrary, a speedy diminution in both the impulse and the size of the aneurism.

While, therefore, the risks accompanying distal compression of the aorta were fully recognised, this mode of treatment might justifiably

be adopted with some hope of success; and more especially because the disease itself was almost invariably fatal in its tendency.

April, 1877.

Malformation of Intestine.

L. MATTHEWS GRIFFITHS.

THE specimen I bring before the Society is the lower part of the intestine of a child who died aged fourteen days without having had any passage through the bowels. When it was four days old I was asked to see it as it had had no motion since its birth, and the ordinary domestic remedies had failed to produce any evacuation. Upon passing the finger just within the sphincter ani a band could be felt extending over the whole calibre of the bowel. This band felt tense, somewhat as it might have felt had it been pressed from above by fæces. Mr. Tibbits, who was kind enough to see the child when I discovered this obstruction, recommended *puncture* of the band with a trocar, and dilatation of the opening by dressing forceps. This I did when the child was six days old, but nothing beyond a small quantity of blood came away. A gum elastic catheter now passed freely, and the finger, now able to pass some inch and a half up the rectum, could detect the edges of the lacerated band. The child from this time became more and more emaciated, refusing food, and died when it was a fortnight old.

Upon dissection it was found that the intestine terminated in a *cul-de-sac* at about the sigmoid flexure, and below this was a mass of fibrous tissue extending downwards for about three-quarters of an inch. It was obvious that it would have been impossible to have safely guided any instrument through this even if the obstruction could have been exactly localised.

Amussat's operation might have been performed, but owing to the uncertainty of the malformation, and the age of the patient, it was considered advisable not to do so.

September, 1877.

Necrosis of Petrous Bone.

J. EWENS.

THE specimen exhibited, a necrosed left temporal bone, was taken from a child at 11 years, admitted into the Hospital for Children. The child had scarlatina seven years previously, and had ever since suffered from otorrhœa and deafness. When admitted there was *complete* paralysis of the left portio dura, a small opening behind the ear communicating with exposed and loose bone; great infiltration with pus of the muscles of the neck and behind the jaw; and the head was drawn to left side. This condition was said to have existed only two or three weeks. Two attempts were made to remove the loose bone, but were attended with profuse hæmorrhage, coming, as proved by post mortem examination, from the lateral sinus.

Child died fourteen days after admission. Death was preceded by marked symptoms of pyæmia, rigors, fluctuating temperature, etc. Consciousness was retained to the last; and the immediate cause of death was exhaustion.

Post mortem examination revealed extensive disease of the petrous portion of the temporal bone, originating in the middle ear, and extending outwards and backwards so as to destroy the structure of the mastoid portion; a large piece of the squamous portion was loose, and the portions of the cerebellum resting on these parts were much stained; and two abscesses, one as large as a small nut, the other smaller, were found in the substance of the cerebellum. The membranes of the brain near this part were much congested, but the brain generally was healthy. A probe passed through the bone directly into the lateral sinus. No further examination of the body could be obtained.

A brother is now and has been an out-patient at the Hospital for some months for otorrhœa of both ears, dating from an attack of scarlatina at the same time.

October, 1877.

Facial Paralysis from Cerebral Tumour.

E. MARKHAM SKERRITT, M.D., M.R.C.P.

THE specimens exhibited were taken from a man aged 42, who was admitted into the Bristol General Hospital on September 9th, 1877, and died on November 5th. There was no history of constitutional syphilis. On admission he had been ill about six months. He first suffered from pain in the head, upon which facial paralysis on the left side gradually supervened. Severe pain in the frontal and temporal regions was still his chief complaint. Left facial paralysis was complete, except that the orbicularis palpebrarum retained slight power, so that the eye could be almost shut. There was complete deafness of left ear; the tongue deviated to the left; and the soft palate and uvula were drawn up to the right. There was no optic neuritis. The patient was rather stupid, and towards the last became a little delirious at night. There were no fits; there was no paralysis of limbs; for although some unsteadiness of gait appeared towards the end of life, this was no more than could be fairly put down to the general failure of strength. There was "inverted re-action" to electricity, *i.e.*, no response to the induced current in the muscles supplied by the left facial nerve, but contraction to the slowly interrupted galvanic current.

Post mortem. New growth was found scattered over the surface of the dura mater as patches of irregular thickening. From the under surface of the right half of the tentorium cerebelli had grown an oval tumour, about one inch long and three quarters of an inch thick, which lay in a hollow on the upper surface of the corresponding lobe of the cerebellum. To the falx cerebri close by, was attached another similar tumour about the size of a pea. In the cerebral hemispheres, just below the surface, were three small tumours about ½-inch in diameter,—one in the left middle transverse frontal convolution; one in the right external inferior frontal; and one in the left inferior temporo-sphenoidal convolu-

tion. The dura mater lining the base of the skull was thickened by patches of new growth; the left facial and auditory nerves were embedded in a nodule which blocked up the mouth of the internal auditory canal. The lungs were infiltrated at their bases with a new growth. In the upper part of the right kidney was a tumour about 1½-inch long and ¾-inch thick, greyish-white on section, and much firmer and less vascular than the cerebral tumours. Microscopical examination showed that these tumours were cancerous,—that in the kidney having the characters of scirrhus, and those in the brain being encephaloid.

In making a diagnosis in this case there were two points to be considered,—the site of the lesion, and its nature. That the lesion was on the peripheral side of the nucleus of the nerve in the floor of the fourth ventricle, and was not cerebral, was shown by the following signs:—first, that there was complete paralysis of the "bilateral" muscles (those that usually act with their fellows of the opposite side, *e.g.*, the occipito-frontalis); and, secondly, by the "inverted re-action" to electricity. It was well known that in lesions *below* the nucleus the paralysis was tolerably uniformly distributed throughout all the muscles supplied by the nerve; while if the lesion was *above*, some muscles were affected much more than others, the *least* affected being those that generally acted with their fellows of the opposite side. These "bilateral" muscles were therefore believed to derive a part of their nervous supply from the hemisphere of the same side. In the present case the paralysis was uniformly distributed, pointing to a lesion on the peripheral side of the nucleus. The partial power in the orbicularis palpebrarum was probably due to the escape of some fibres of the nerve from destruction by the new growth. Again, with regard to the electrical reaction. In lesions *above* the nucleus there was only slight increase or slight diminution in irritability of the affected muscles; and the same change to both forms of electricity; while in lesions *below* the nucleus there was very marked "inverted reaction," *i.e.*, diminished irritability to faradisation, increased

irritability to galvanism,—the former being due to the fact that the nerve itself underwent degeneration in consequence of being cut off from the nucleus which governed its nutrition, while the latter was the reaction of the muscles themselves. In this case there was this "inverted reaction." That the lesion was "basilar"—at the base of the brain—rather than in the after course of the nerve, was indicated by the associated paralysis of the auditory and hypoglossal nerves.

As regarded the nature of the lesion, the history and course of the case pointed to chronic meningitis, simple or syphilitic, or tumour.

It was to be noticed that there were no distinct symptoms referable to the cerebellum, in spite of the compression of the right lobe by the tumour growing from the tentorium cerebelli.

The tumour in the kidney was probably the primary growth, as it was single and had the characters of scirrhus, while both in the lungs and in the brain the growths were multiple and encephaloid in structure. The brain was probably infected from the secondary deposits in the lungs.

November, 1877.

Sub-cutaneous Emphysema and Infiltration with Pus following Aspiration for Pyo-pneumothorax.

E. Markham Skerritt, M.D., M.R.C.P.

THERE were two points of special interest in this case,—the mode of production of the pyo-pneumothorax, and the rare and probably unique accident following aspiration.

The patient was a young woman, aged 23, admitted into the Bristol General Hospital with a history of cough and dyspnœa for some months; and three days before admission rigors and pain in the shoulders and back. When seen on Dec. 11th, 1877, she was lying propped up in bed, breathing very rapidly and with difficulty.

The respirations were 64, the pulse 140 and very weak; the temperature the night before was 104°, and in the morning, 100·8°. There were the physical signs of pyo-pneumothorax on the left side; at the right base abundant rales, and also pleuritic friction. As there was great dyspnœa, the aspirator-trochar was inserted in the ninth interspace, in a line with the angle of the scapula, and three and a quarter pints of very fœtid thin pus were drawn off, and also a quantity of most offensive gas. The dyspnœa was but little relieved. On the second day after, the patient was much worse: the breathing was laboured and hurried; the surface cold, and covered with a clammy sweat. When moved she complained of tenderness over the left side, especially towards the groin. The skin round the aspirator puncture, over an area of about four inches, was of a bluish colour and puffy, and on applying the hand the peculiar crackling of sub-cutaneous emphysema was felt. This emphysema extended over both sides, as high as the angle of the scapula, forwards to the posterior axillary line, and downwards to the iliac crest. In the left loin the skin was red and brawny. On auscultation, very coarse bubbling rales were heard over the skin around the puncture. As the contents of the pleura were known to be decomposed, only one result of their extravasation into the tissues could be expected,—suppuration all along their track, such as occurs on extravasation of urine. It was plain that there was but one course open,—to allow the pus and gas to escape freely externally instead of into the planes of areolar tissue. Accordingly a drainage tube was inserted at the site of the former puncture. However, the patient's condition did not improve, and she died from exhaustion about six hours after.

Post mortem examination sixteen hours after death.

When the skin over the pubes was cut through, fœtid gas escaped with a hissing sound. The whole abdominal wall on the left side, and the whole region over which emphysema was detected during life was more or less infiltrated with gas and fœtid pus. The left lung was shrunken and carnified, and was covered and

bound down by a thick layer of lymph, evidently of old standing. No trace of phthisis, rupture, or other disease (apart from the effects of compression) could be found in the lung. The right lung was emphysematous and intensely congested; over its base was some recent lymph.

What was the source of the gas in the pleural cavity? The common cause of pneumothorax was perforation of the cavity from without, most often by the giving way of the pleura over a phthisical deposit in the lung; the symptoms then were generally severe and sudden in their onset. But here there was no such sudden attack. The patient about a year before had had some acute chest affection, and since had suffered from cough and dyspnœa. It is true that three days before admission an acute febrile attack set in, but that was not accompanied by the usual symptoms of perforation, and was considered due to the pleurisy that had appeared on the other side. So that the history seemed to indicate a gradual production of the pyo-pneumothorax; and the *post mortem* evidence agreed with this; for the lung had been collapsed for a long time, and there was no evidence that there had been perforation from it. The lung was apparently perfectly healthy before it became compressed. If, then, no cause external to the pleura could be found, might not the pus have originated in the cavity itself from decomposition in an old empyema? As regarded the possibility of such decomposition, authorities differed widely; for while most English writers recognised it as a rare occurrence, Fraentzel, in *Ziemssen's Cyclopædia*, says, "No distinct proof has ever been adduced of the origin of pneumothorax in this way; indeed, it appears clear from the researches of Ewald that no development of gas can take place in such a manner." Notwithstanding this, the author was disposed to regard this case as one in which the gas did result from decomposition in an old empyema without access of external air. It was known that decomposition might take place in closed cavities, and here there was no evidence of any other source; while the history

of the case and the *post mortem* evidence alike pointed to a chronic course and a gradual accumulation of gas.

Next with regard to the unfortunate sequel to the use of the aspirator: extensive infiltration of the pleural contents into the sub-cutaneous tissue through the opening made by the aspirator trochar, usually so harmless; this was an accident which, so far as the author could discover, had not before been met with. As the result showed, it would have been best to have opened the pleural cavity freely at first, but the complication that occurred was one that had not hitherto been taken into consideration. When once this infiltration had taken place, there was evidently but one course open,—to provide a way of escape externally for the fœtid pleural contents. In a similar case, if the author decided on aspiration, he would be disposed to watch narrowly the site of puncture, and make a free opening into the pleural cavity if there was any evidence that the contents were passing into the sub-cutaneous tissue.

Although death was to be attributed immediately to the unfortunate accident, yet if the patient had been left alone she must inevitably have died, and that probably at no later period than was actually the case.

January, 1878.

Hæmorrhage into Pons and Medulla.

DR. J. GREIG SMITH.

DRAWINGS of a specimen of punctiform hæmorrhages into the medulla oblongata, and a specimen of hæmorrhage into the pons and medulla were exhibited. Both were from cases that had died within the year at the Bristol Royal Infirmary.

The first case (of small hæmorrhages) was profoundly comatose when seen, and death was found to have been caused by a large

hæmorrhage into the right corpus striatum. In a section through the middle of the medulla were six distinct hæmorrhages, confined to the posterior half of the body, of sizes varying from the head of a pin to a small pea. They were of recent origin. The cerebral vessels were atheromatous, and there was cardiac hypertrophy and cirrhosis of kidneys. The patient was a male, aged 49.

The other case, of hæmorrhage into the pons and medulla, was from a man aged 48. He continued sensible for two hours before coma supervened, and died six hours after the attack.

When first seen the patient was sedulously calling the attention of bystanders to the fact that he could not close his left eye. With his left hand he kept pointing alternately to his left and to his right eye, showing how he could close one but not the other, and made a graphic dumb display of his condition. On the left side of the face there were the other ordinary symptoms of paralysis of the facial nerve.

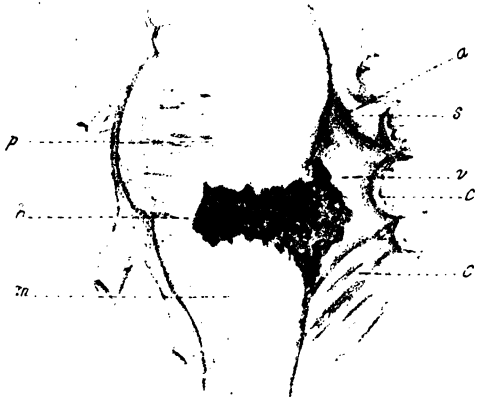
The limbs on the right side were continually fidgetting, and he made repeated but futile endeavours to flex them. When his right arm fell over the stretcher on which he was lying he was unable to bring it up again though he made strenuous attempts to do so; but, when the forearm was flexed on the arm, and the limb laid over the chest, he did not seek to move it. Precisely a similar state of affairs existed with the right leg; ineffectually endeavouring to flex it while in the extended position, he permitted it to rest after it had been flexed for him. It was evident enough that he could not speak for he continually attempted to do so. He appeared also to be perfectly deaf. There was no strabismus, and both eyes moved with parallel axes in every direction. There was marked and equal contraction of both pupils. He swallowed some water with little difficulty. So far as could be made out—for it was impossible to induce him to move it—there was no paralysis of the tongue. There seemed to be no loss of sensation anywhere—on face, mucous membrane of mouth, or conjunctiva. The soft palate was not affected. The left side of the body and the right side of the face were unaffected.

DESCRIPTION OF PLATE IX.

Plate IX. Illustrating Dr. J. Greig Smith's case of Hæmorrhage into pons varolii and medulla oblongata.

p.—Pons Varolii.
m —Medulla Oblongata.
b.—Blood clot.
c. c.—Cerebellum.
a. —Entrance into Aqueduct of Sylvius
v.—Cavity of fourth ventricle.
s.—Superior peduncle of cerebellum.

Plate IX



by Dr J. Greig Smith

The post mortem examination revealed extreme emphysema of both lungs, general cardiac hypertrophy (heart weighed 14oz.), atheroma of aorta, and very advanced atheromatous disease of all the cerebral vessels. The fourth ventricle was distended with dark clotted blood, and there was a deep transverse rent in the nerve tissue between pons and medulla, and some superficial laceration of the structures on the floor of the fourth ventricle. The blood had not got beyond the cavity of the ventricle. (*See plate.*)

Having commented on the rarity of hæmorrhage into the medulla, Dr. Greig Smith called attention to some points of interest in his second case, such as the escape of the sixth nerve while the seventh was implicated (both having a common nucleus), and the length of time for which the lesion remained unilateral and the primary symptoms stationary.

Discussing the possible causes of the contraction of pupil commonly seen in hæmorrhage into the fourth ventricle, he concluded that it depended on irritation of the grey nucleus of the third nerve in the floor of the aqueduct of Sylvius; the blood forcing its way along that canal, and causing compression or laceration of the structures surrounding it.

January, 1878.

Syphiloma of the Heart.

DR. SHINGLETON SMITH.

THE author exhibited a heart, showing an almost unique condition of syphiloma, at the meeting on January 23rd, 1878. The patient from whose body the heart was taken was a woman aged about 35. She had fallen dead suddenly in the street, and no history of previous illness could be elicited. No disease was found in any organ except the heart. The greater part of the heart's texture was infiltrated with a morbid growth, but very unequally

so. On the surface of the left ventricle pale coloured nodules were visible, projecting above the surrounding tissue, and extending to a depth of half an inch into the heart's texture. Projecting internally were many other nodules as large as peas, growing on the muscular columns. The greater portion of the inter-ventricular septum was uniformly infiltrated, being pale in colour and containing very little muscle structure.

Microscopically the nodules were found to consist of a coarse fibrous stroma infiltrated with a small-celled growth; elsewhere the small-celled growth was found to infiltrate muscle structure running in lines between the fibres, and collected into masses here and there. The muscular tissue itself was granular; many of the fibres had wasted, and the inter-muscular septa were generally thickened. The adipose tissue on the heart's surface was similarly infiltrated, lines of minute nuclei being seen to run between the cells, and to be grouped here and there in larger masses.

No evidence of syphilis was noticed in the body. The characters of the morbid growth formed in the ventricular walls agreed only with those of syphiloma. The condition was not one of fibroid change. There was no tendency to caseation, nor to the development of secondary aneurisms.

January, 1878.

Spina Bifida.

J. EWENS.

MR. EWENS exhibited a child cured of Lumbar Spina Bifida, well illustrating the advantage of iodo-glycerine injection. The child was born February 11th, 1877; was first seen by Mr. Ewens in consultation February 21st. The tumour was rather larger than a large walnut, and rapidly increasing; coverings thin, but not transparent. Child otherwise healthy, but rather small.

DESCRIPTION OF PLATE X.

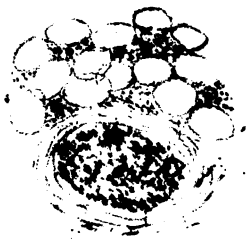
Plate X. illustrates Dr. Shingleton Smith's specimen of Syphiloma of the Heart.

FIG. 1.—Small vessel and adipose tissue at the surface of the heart, shewing infiltration of the connective tissue with minute nuclei.

FIG. 2.—Thickening of the inter-muscular stroma, and infiltration with nuclei.

FIG. 3.—A small nodule invading the muscular structure, running between and separating the fibres.

Drawn by R. Shingleton Smith.



x 70.

Fig. 1.

x 240.

Fig. 2.

x 140.

Fig. 3.

On March 6th preliminary tapping was performed, and one or two drachms of clear fluid were drawn off. No effect on the child was perceptible. Wound closed by collodion.

March 16th. Three drachms of fluid drawn off, and m xv. of iodi-glycerine injected, (gr. x of iodine and gr. xxx of potas. iodi. ad. ʒ iij of glycerine.) Child looked pale after operation. Puncture was closed with collodion, and a pad of lint. Convulsions occurred three hours after operation, and recurred with more or less severity and frequency for ten days, when they passed off. The child was almost paraplegic. Obstinate constipation and retention of urine followed. Catheterism was necessary on March 27th, and as phymosis existed, this was operated on a few days after.

From this time till October the child was in a very fluctuating condition, emaciation and general debility occurring. A severe attack of whooping cough seriously interfered with recovery.

The treatment most useful for the case generally, with special reference to the constipation, was a combination of potas. bromid. and belladonna.

During the period of greatest emaciation a floating left kidney could be distinctly felt. The swelling never returned after the puncture, the skin gradually thickened, and so far the Spina Bifida was cured very soon, but the constitutional symptoms were for seven months of a nature to excite anxiety. In October matters had reached a very bad condition, calling for more regular and energetic treatment, which had been rather neglected.

℞ Potas. Iodi. gr. ½, Potas. bromid. gr. ij. Tr. belladonnæ m. iii. ter. die.

Ung. potas. iodi. rubbed into the abdomen, and cod liver oil rubbed all over the body twice a day, effected a speedy and regular improvement. The child is now well-nourished, and has perfect use of legs. Some thickened skin alone remains to mark the position of the Spina Bifida.

Mr. Ewens also mentioned a case which occurred in his practice at the Children's Hospital in 1876.

A child six weeks old was brought to the out-patient department with a large Lumbar Spina Bifida. Three injections of iodi-glycerine solution were used. Convulsions followed, but gradually yielded to potas. bromid. and belladonna. The child went on well for two months; the skin was gradually thickening over the tumour which was reduced to a very small size, when unfortunately an attack of measles occurred: acute hydrocephalus supervened, and the child soon died, about three months after being first seen. The progress of this case was so satisfactory that a perfect cure appeared likely. No post mortem examination could be obtained.

The mother was a very highly strumous and delicate woman and the child had a head rather larger than usual, and the general physique was unhealthy, so that the case was not a promising one from the commencement, hence the amount of success achieved was satisfactory, and may be claimed in favour of the iodo-glycerine treatment. As the head enlarged the spinal tumour again increased and the skin got very thin.

February, 1878.

Case of Varicose Aneurism of the Left Orbit, cured by Ligature.

F. Poole Lansdown.

My patient Mr. H. was wounded at the inner side of the left upper eyelid, by the bursting of a soda water bottle while he was in the act of stooping and opening a hamper. Being on the spot I saw him at once. The wound penetrated the upper lid; it was about half an inch in length. The eyelids were distended to their utmost by effusion of blood into the cellular tissue, and an artery was jetting blood from the wound. I brought the edges of the wound together with a suture which stopped the bleeding, and

in a few days he was apparently well. In about six weeks, however, the eye gradually became prominent, the eyelids swollen and the veins of the conjunction tortuous and distended. In a few weeks a small pulsating tumour was discovered at the inner angle of the orbit, beneath the cicatrix of the old wound. There was a distinct murmur heard over this, as well as over the eye. The sight was perfect; rest was enjoined and remedies used to restrain the circulation; pressure was applied to the little aneurism by means of a truss, with the idea of compressing it against the inner wall of the orbit; but as this gave rise to much pain, and pressed the tumour into the orbit rather than arrested the pulsation, it was discontinued.

About this time he saw Mr. Bader and Mr. Higgins of Guy's Hospital, and from the fact of the retinal veins being as dilated as the superficial veins, Mr. Bader was of opinion that there was an aneurism deep in the orbit and advised ligature of the carotid. I prefered trying an exploratory operation; accordingly, on February 18th, 1874, I cut down and opened up the old wound exposing the small globular pulsating tumour, with a large tortuous vein coming off from the front of the sac, and passing to the back of the orbit. Between the eye and the aneurism there were several smaller vessels going to and surrounding the sac, which made the operation tedious. Having found the feeding vessel, I placed a carbolised ligature on the cardiac and distal sides of the aneurism, and closed the wound; on the fourth day the sac was discharged, in a week the eye had returned to its natural level, and my patient made a steady and complete recovery.

This case is interesting, showing that an aneurism communicating with the opthalmic vein may simulate a deep seated aneurism in the orbit, producing great engorgement of all the veins in the orbit with considerable exopthalmos.

The patient is now, 1878, quite well, the eyes being symmetrical.

November, 1874.

On Conjoined Epithelium.

THE LATE S. MARTYN, M.D., F.R.C.P.

At a Meeting held on March 24th, 1875, Dr. Martyn (then Vice-President of the Society), gave a demonstration of and read a paper on "The Prickle-cells found in Rodent Ulcers." The following paper, reprinted from the British Medical Journal of June 26th, 1875, is given in full as the latest scientific work of our late President.

IT does not appear that much attention has been given by English observers to the so-called "prickle and ridge" cells (*Stachel-und Riffzellen*). At all events, they have scarcely found their way into our most recent books on pathology; while the drawing in our newest text-book on minute anatomy is almost if not quite, identical with the earliest sketch in Virchow's *Archiv*.

It is, nevertheless, twelve years since these cells were first discovered and named by Professor Max Schultze; and their distribution in fishes and amphibia has been fully described (1867) by his brother, Professor Franz Schultze. They have been subsequently noticed by Kölliker, Frey, Rindfleisch, Stricker, Cornil and Ranvier, Rollett, and others. They have been called "spinous cells," "echinate cells," "cellules dentelées," "ribbed" and "spiny and furrowed" cells; but no observer seems to have added any careful researches to the original description of Max Schultze, by whom the name was given to them of "prickle and ridge" cells. And yet there is a certain beauty and interest attaching to these structures which seem to entitle them to more detailed notice; and, while they offer problems in histogenesis of great interest to the anatomist and physiologist, they cannot but do so also in some degree to the pathologist. It is a good illustration of the old story of "eyes and no eyes," that a very remarkable structure, and not a difficult one either, in so thoroughly

common a place as the borders of the epithelial cell, should have been persistently overlooked for many years, and long after high microscopic object-glasses had been brought to great perfection. No one need despair of seeing something new and important by looking with extra carefulness at what is, so to speak, straight before his face, after this fact, that the mere form of the simplest of all animal cells actually remained unnoticed until a dozen years ago.

The discovery of Max Schultze, then, was this; that in the epithelium of the mouth and conjunctiva firstly, and then in the epidermis of the skin, and indeed wherever there is true pavement or scaly epithelium in many layers, the second series upwards of cells have this peculiar character. Their surface is furnished with short projections, which are apt to appear as prickles; while sometimes there are ridges (reefs) marking portions of the cell with parallel groovings (see drawing). The lowest layer of epidermic cells consists of elongated vertical palisading-like elements, between and underneath which lie germinal masses of indeterminate form. Next above, and completing what was called "rete mucosum," is a succession of layers of spheroidal cells, becoming more and more flattened until they cohere under the form of mere scales into cuticle on the skin, or in a less firm manner in transitional situations like the mouth, conjunctiva, etc. It is these latter layers which are mostly spinous. There is some difficulty in seeing this well in thick skin. It was already noticed by Max Schultze, that the diseases involving the true skin showed the prickle-cells well; and other observers, especially Cornil and Ranvier, have followed in the same line (Fig. 1.)

The anatomical structure, then, has been described thus: the cells are covered more or less with spines, which, when the cells are isolated, show as marginal prickles; and if the covering pressure chance to be *nil*, as besetting the surface. The spines are sharp-pointed, with a broadish base; and all observers, I believe, describe them as interlocking with the teeth of the next cells, thus giving the characteristic cohesion. Max Schultze

compares this to the intermingled bristles of two brushes put together. Ranvier speaks of the cells as soldered by the interlocking teeth ("les dentelures au moyen desquelles elles sont engrenées et soudées"). Stricker uses the same phrase, and Rindfleisch compares their union to "a suture"; and I learn that the well known Edingburgh *preparateur* Mr. Stirling, discovering them nine years ago, thought them like "watch-wheels" (see Fig. 1.)

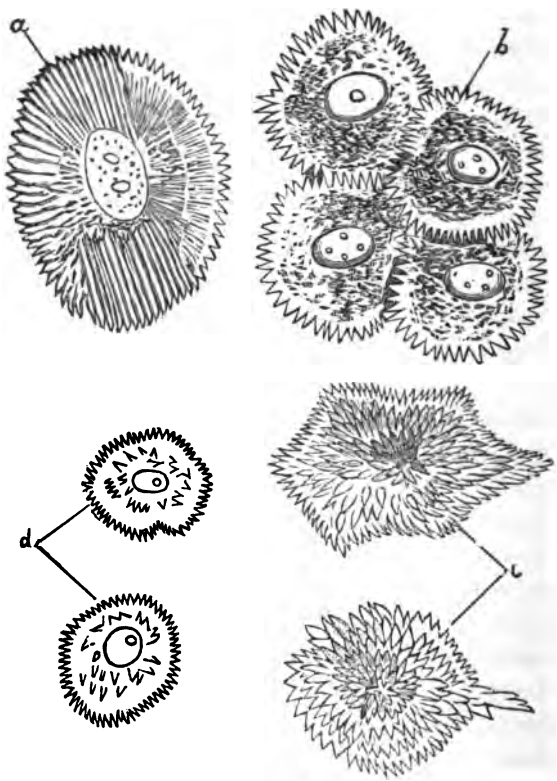


Fig. 1.—Epidermic "Prickle and Ridge" Cells: or "Spinous," "Furrowed," or "Echinate."—*a*. Papillary Tumour of the Tongue. *b*. Human Epidermis (Max Schultze). *c*. Ext. Layer, Middle Cells—Cornea of Pig: Stricker and Rollett. *d*. Cellules dentelées of Cylindroma: Cornil and Ranvier (1869).

At first sight, this all seems satisfactory enough; but it is just as to the simple nature and plan of these processes that I venture to join issue with all observers to whose works I have been able to obtain access. So long, namely, as I looked at the contact between any two of these cells as that of toothed wheels, they really seemed to interlock. I confess, however to a rooted distrust of real processes of definite form as being projected from a cell-wall of formed material, this having no analogy with the many changes of form in germinal or bioplastic matter. As far back as 1860, when the language of, I believe, all the books was of processes sent out by cells, I ventured to dissent from such views, and, in a paper on Connective Tissue published in Beale's *Archives*, described cell processes as being "spun out" by a receding body. This view is now generally accepted. It seemed, therefore, to me worth inquiry, whether these prickles were really processes which interlock, or whether they were really continuous delicate bands uniting cell to cell. In an epithelioma of the lip, Fig. 2, and in a section of a hard preputial chancre, I found instances of a rich development of the prickle-cell. Probably, just as on a nasal polypus the ciliated epithelium often becomes monstrous, many fine examples of these cells may be encountered where there is unusual setting free of formative force.

In the example (Fig. 2), the cell (*a*) is incontestably united by bands to its neighbours, and the decisive experiment has been made; for, where these bands have broken across, the remaining stumps have become prickles; but where (two cells touching) the teeth seem to have strongly dark ends (Fig. 2, *b*), I believe this effect is universally an optical illusion, and that the dark spot is the cavern between bands (*b*). On the other hand, where (two cells touching) the ordinary *light* spines seem to be so truncated a very careful view will show that the light track may be followed from one cell to the next, if I may use the illustration, like walking across on a plank. In some cases, however, pointed teeth may be

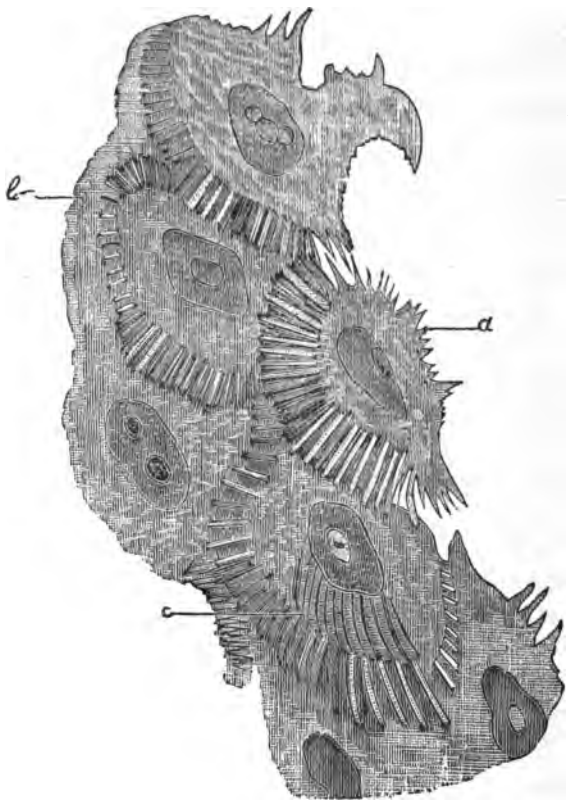


Fig. 2.—Conjoined Epithelium—1,000 Diameters.

seen as touching cell-margins, as in Max Schultze's original plate. But it is an illusion, in my opinion, to see in this appearance a true second set of interlocking teeth, the outlines of which correspond precisely with the first. Rather let it be granted that prickles are broken bands of union in all cases, as we know they are in (*e.g.*, *a* Fig, 2,), and then the whole thing becomes intelligible. In a microscopic preparation thin enough to be seen well with 1,000 diameters, most cells are partly isolated, and, consequently, broken

bands, *i.e.*, prickles, abound and those near the outer margin of the cells lie as detached teeth touching from each cell-wall, or even interlocking in some instances. Meanwhile, the cell-margins of real contact deeper down are united by unbroken bands.

So much for the prickles; and next as to the "furrows," "reefs," "ribs," or "ridges" (see Fig. 1. *a*, and Fig. 2, *c*). I confess that at present the only explanation which occurs to me is founded on an insufficient number of observations. As a conjecture supported by some instances, I would suggest that these are *stretched bands* often broken off at one end, and lying parallel on the cell-wall. They are beautifully seen with careful oblique illumination under high powers.

This peculiar cell-structure which we have been describing may, then, be supposed to originate in this sort of way. In the lowest cell-layer of epithelium, there are found the newly grown, long, vertically placed cells, side by side in contact. When these have a somewhat permanent character, as in many fishes, the next layer of cells consists of spheroidal cell-forms which have resulted from division or budding, and consequent "fissiparous multiplication" or budding of a *new* progeny. In all this there is a tendency of dividing cells to remain, at points, united by threads of formed material of hard cell-wall; *e.g.*, amongst these very layers are often found large and branched pigment-cells united by long delicate threads. Thus I imagine the cells of rete mucosum, in multiplying originally by subdivision, retain numerous points of incomplete severance, and these points of cohesion are dragged out and become the uniting bands. These, when severed by any accident, assume the characteristic form of "prickles." In the normal ascending series, these cells, becoming older and flatter, lose all surface-connection, and in the cuticle are simple polyhedral horny blocks. The uniting agency of these cells is a firm one; for in a scraping from a tumour-section, as noticed by Ranvier, they almost always hold together strongly in groups. It is a firmer union than I should expect from any interlocking teeth, but of course one which would naturally result from uniting bands.

Lastly, as to the pathological anatomy. The primary meaning of the presence of these cells is an abnormal formative activity of the lower cells in the rete mucosum; and the occurrence of rib and prickle cells may be taken as indicative of this structure having become involved. In epithelioma occuring in connection with *flattened* epithelium, both lobular and tubular, they mostly occur. Their development seems to be in direct ratio to the vigour of growth present, and accordingly they were very large in granulations snipped from a perineal fistula. Papilloma and rodent ulcer afford fine specimens; and probably always the best occur in diseases of of the transitional epithelium extending from the lips to the cardiac orifice, and on the conjunctival, anal, or preputial epidermis. I have found them unusually fine in a section of preputial chancre. I believe it to be the opinion of Professor Lister, who discovered these cells independently, and name them "echinate," that rodent ulcer, when they are present in it, should be considered as allied to, if not identical with, epithelioma. The presence of prickle-cells in cylindroma leads me strongly to doubt the origin of that form of cancer from the sudoriparous glands, according to the received notion. But as I indicated at first, the real meaning, for medicine and surgery, of these curious cells, remains to be investigated; and it is necessary that some one should do that for their origin, varied forms, monstrosities, and pathological distribution which has been so laboriously accomplished for their anatomy in fishes and amphibia by Professor F. Schultze. My present attempt has been, so far, merely to see more of the true character of their intimate structure, and, if possible, to determine the true mode in which they are united together.*

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* Four situations may be recommended specially to those who wish to study these cells. 1. The lip of the sturgeon. 2. The cornea of the pig. 3. Preputial chancre. 4. Epithelioma of the lip.

DEBATE

ON THE

TREATMENT OF HIGH TEMPERATURE OF THE BODY.

THIS debate was opened by Dr. SHINGLETON SMITH on Wednesday, May 23rd, 1877.

The first question considered was, *Does Pyrexia require treatment,* and if so, within what limits is treatment necessary?

The dangers of high temperature *(per se)* are :—

1.—Coagulation of heart myosin at a temperature of 110° Fahrenheit.

2.—Granular degeneration of the muscles and parenchymatous tissues generally.

That this degeneration results from the pyrexia is shewn by—

(a)—Experiment on animals. The observations of Schultze and Kühne on the vitality of protoplasm, and those of Wickham Legge on rabbits, illustrate this point.

(b)—The constancy of the anatomical lesions in the various diseases in which pyrexia is a constant symptom. The similarity of the lesions to those found in fatal cases of heat-fever indicates that the elevation of the body temperature is probably the exciting agent of tissue degeneration.

(*c*)—Clinical experience indicates that the danger to life varies with the degree of the pyrexia and its duration. These two factors must both be considered. A temperature above 104°, and even up to 107° for a short period, need not give rise to tissue change; whereas a lower temperature, of say 103°, when protracted becomes dangerous.

(*d*)—The results of anti-pyretic treatment indicate that a systematic reduction of the temperature gives rise to a diminished mortality.

The next question considered was, *By what methods* can the temperature be reduced? The various methods for the reduction of temperature were reviewed, and temperature charts were shewn illustrating the effects of different agents. The cold bath and its modifications were stated to be the most efficient and ready means at our disposal in all cases where the immediate reduction of temperature is necessary. Quinine ranks next as the most powerful anti-pyretic agent, and charts were shewn illustrating the effect of large doses of quinine on the pyrexia of pneumonia and enteric fever. The quinine was given in the way recommended by Liebermeister: a full dose of thirty or forty grains being given within an hour in divided doses at intervals of ten or fifteen minutes.

The following is a summary of the results.—*Quinine:* dose of forty grains for an adult man.

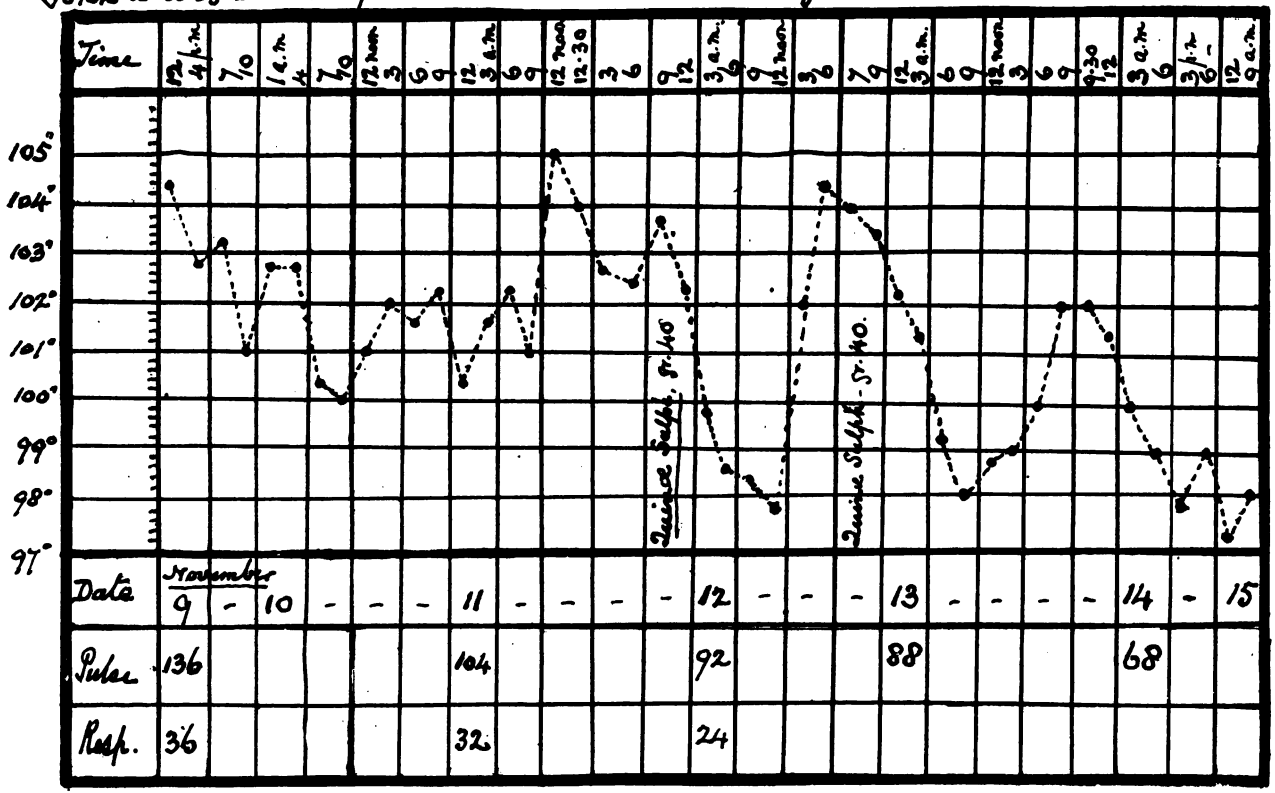
AVERAGES OF SIXTEEN CASES.

Temperature for which given	104·3	degrees.
Highest	105·4	,,
Lowest	103·4	,,
Amount of fall	5·6	,,
Highest	10·0	,,
Least	4·4	,,
Time in falling	12	hours.
Longest	24	,,
Shortest	4	,,

DESCRIPTION OF CHART I.

Chart I. illustrates the effect of quinine in the reduction of temperature during the course of acute pneumonia. On the third day after admission the temperature having been as high as 105° Fahr. a dose of forty grains of quinine was given at nine o'clock p.m., the temperature then being $103\frac{3}{5}$. On the following day at twelve noon, fifteen hours after the quinine was administered, the temperature had fallen to $97\frac{3}{5}$; it rose again on the same evening to $104\frac{2}{5}$, when a second (gr. xl.) dose of quinine was given; fourteen hours later the temperature had again fallen to 98°. After this there was no further considerable rise, and the patient made a speedy recovery.

John Davis - Æt. 27. Pneumonia - Recovery.



R.S.S.

Chart I.

DESCRIPTION OF CHART II.

Chart II. Also a case of acute pneumonia. The temperature ran a high course, and the general symptoms were correspondingly severe until a dose of quinine (thirty grains) was given on the evening of July 31st. Profuse perspiration occurred during the night, and on the following morning at nine o'clock the temperature was as low as 94°; it continued below 96° during a period of six hours, and then rapidly ran up again to 105⅘. No further doses were given, as a steady fall took place, and there was subsequently no protracted rise. Convalescence was brief and uninterrupted.

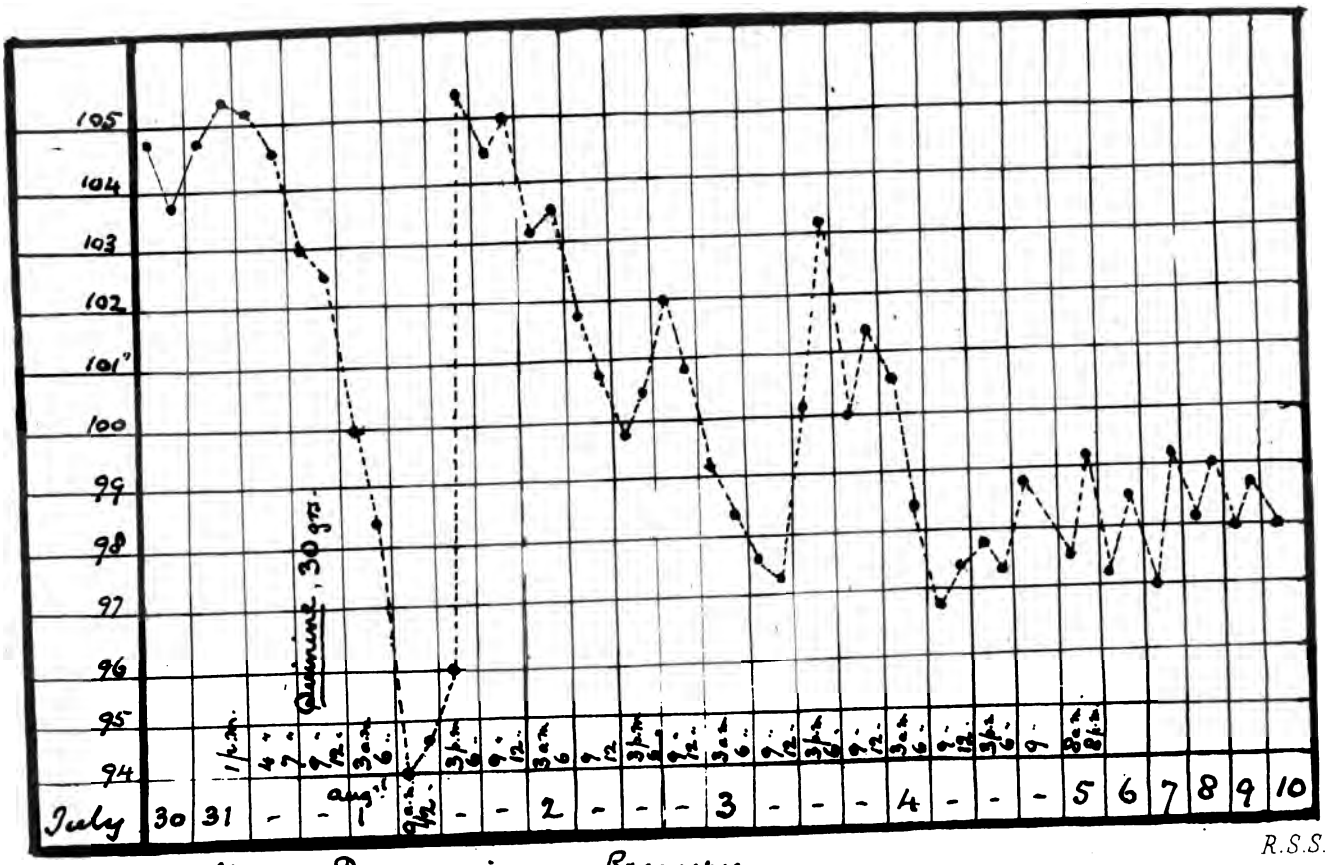


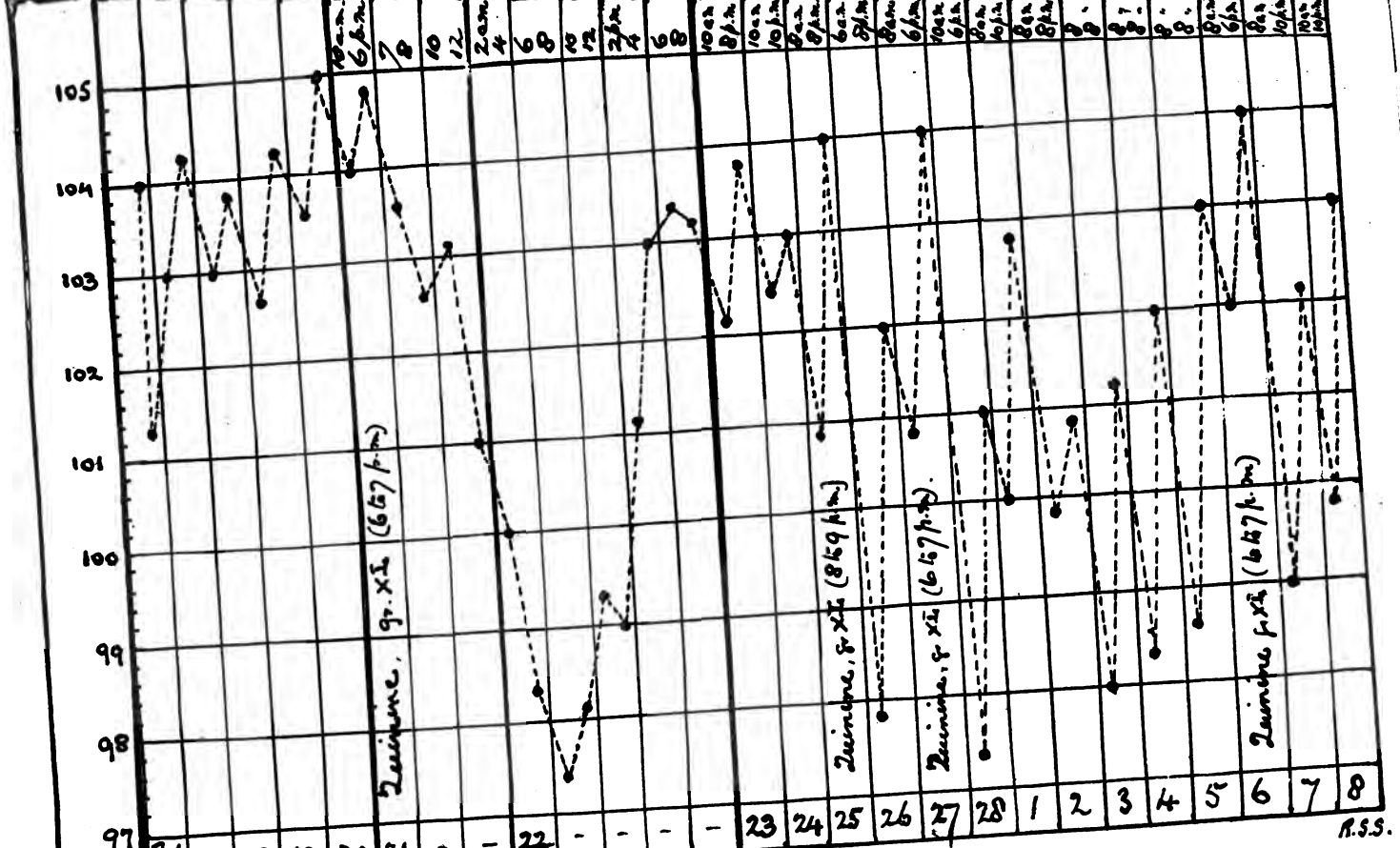
Chart II

R.S.S.

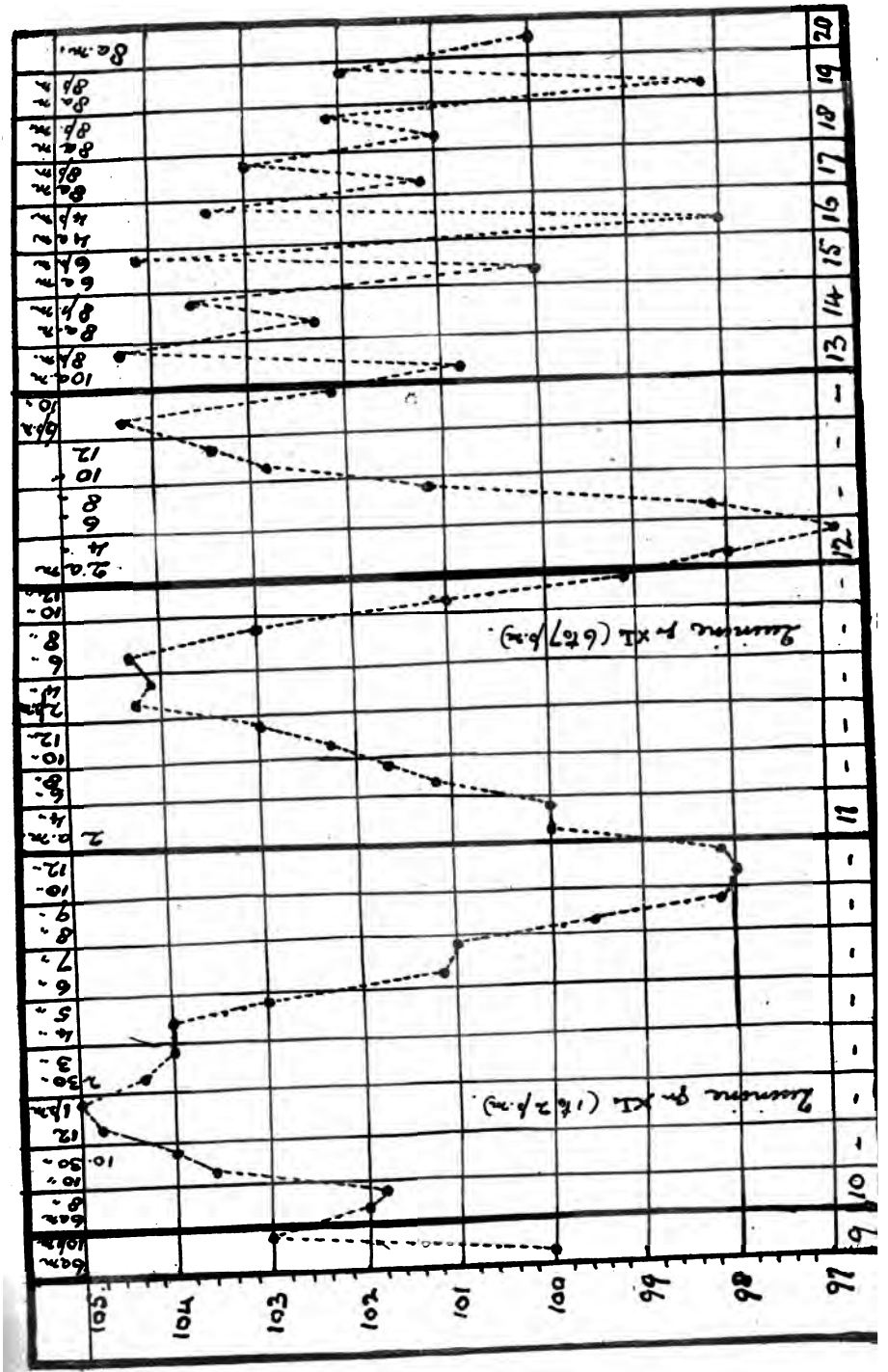
F.H. Ch. 14 - Pneumonia Recovery.

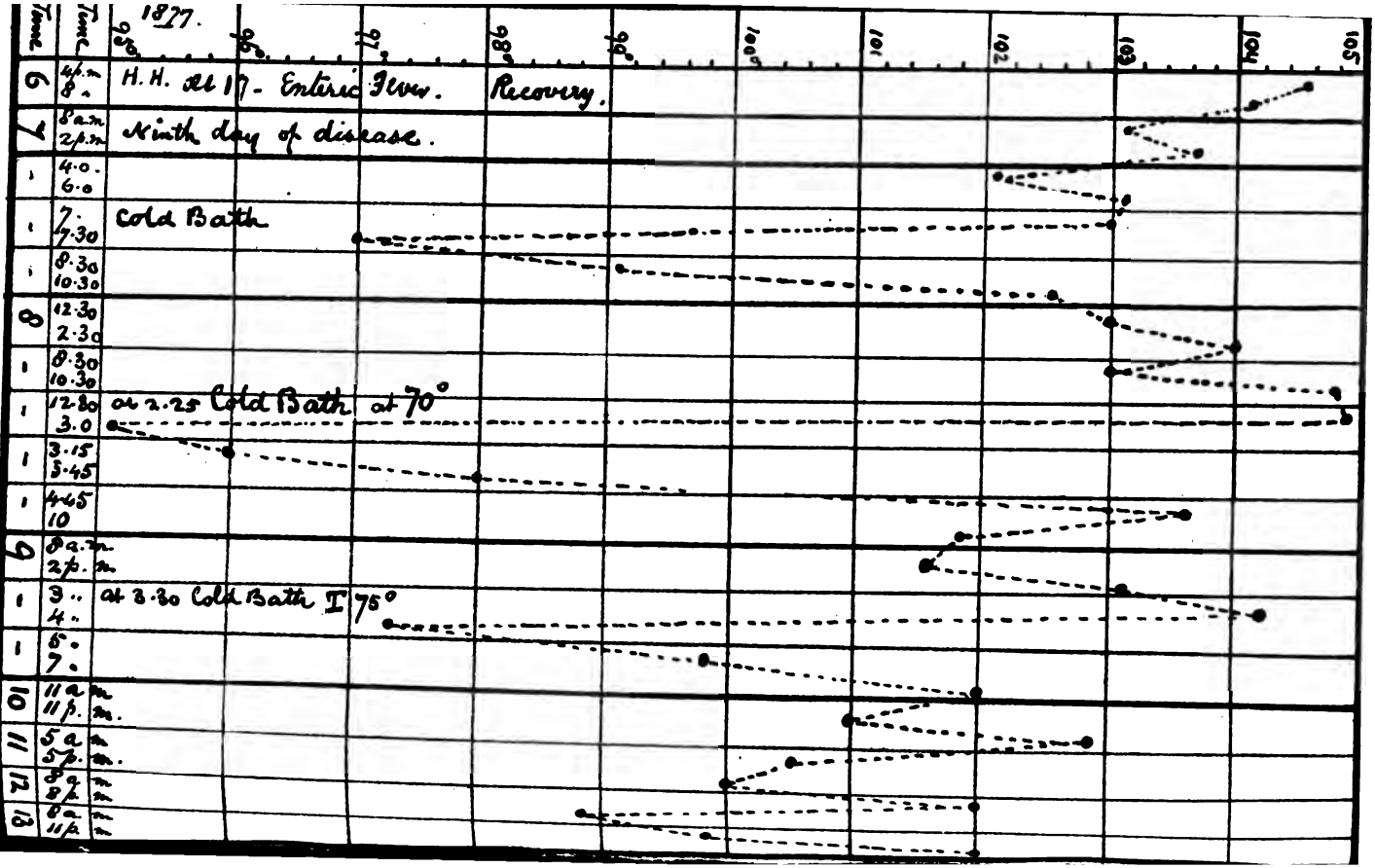
DESCRIPTION OF CHARTS III., AND III. CONTINUED.

Charts III., and III. continued. These charts illustrate the effect of quinine in the treatment of enteric fever. It was an unusually severe case of the disease occurring in a robust previously healthy policeman. As many as six doses of quinine of forty grains each were given during the course of the primary attack and the relapse, but no other anti-pyretic treatment was adopted. The results of the first, fifth and sixth administrations of the drug are indicated in the charts in detail; that of the others is epitomised. Stimulants were given in large quantities (twelve ounces of brandy in twenty-four hours), but did not appear to influence the temperature. The heart's action had been weak from the commencement, but it always improved under the influence of the quinine. The patient made a very slow recovery, and for weeks had a very rapid, small, and weak pulse, with œdema of the legs, but recovery was ultimately complete.



Temperature - Ot 22. Enteric Fever - Recovery, after relapse -


Summer gn XI (1&2 p.m.)
Summer pn XI (6&7 p.m.)

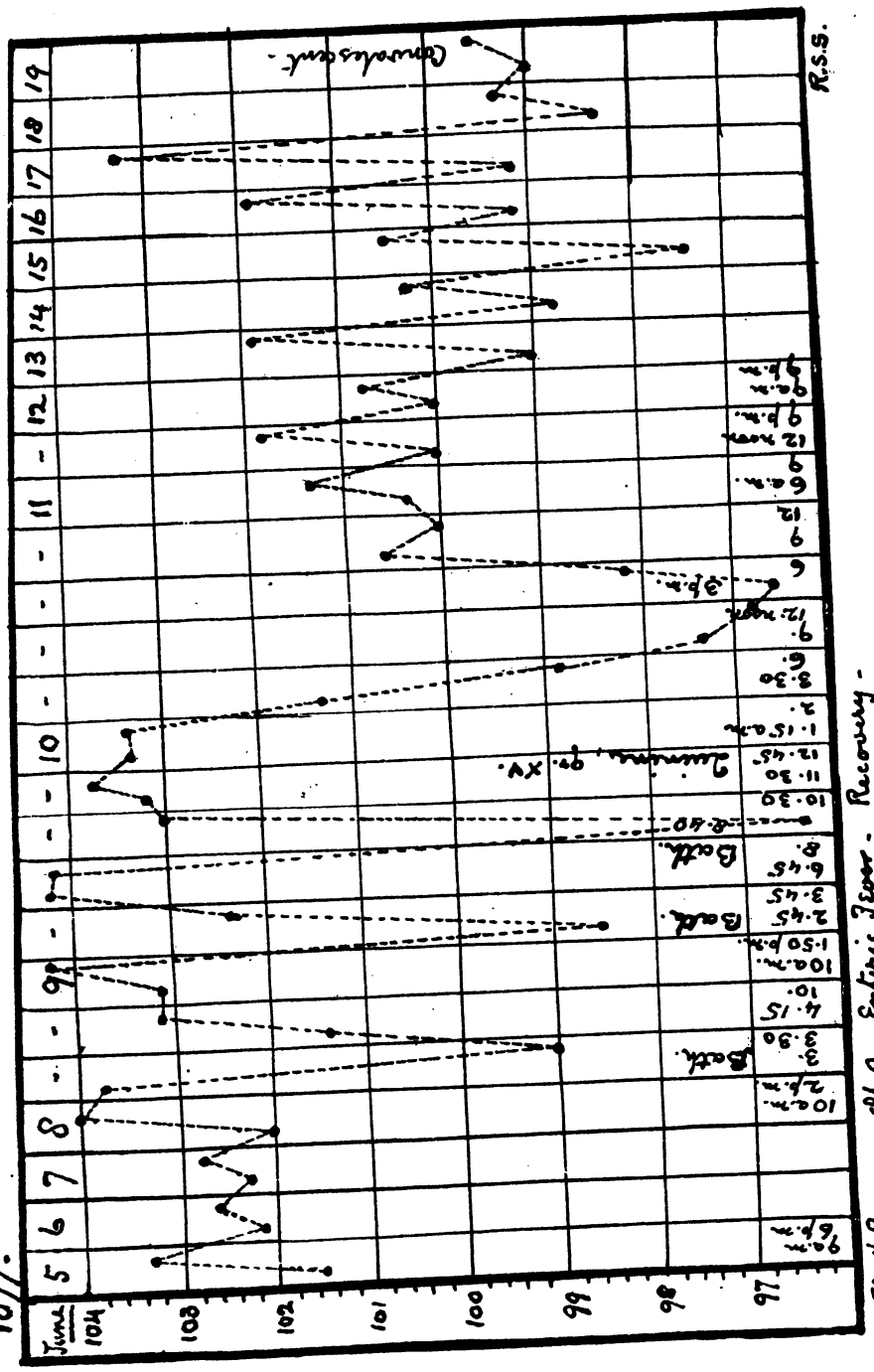

1877.
Time
Time
96°
97°
98°
99°
100°
101
102
103
104
105
6
4 p.m.
8 "
H. H. Æt 17 - Enteric Fever.
Recovery.
7
8 a.m.
2 p.m.
Ninth day of disease.
1
4.0.
6.0
1
7.
7.30
Cold Bath
1
8.30
10.30
8
12.30
2.30
1
8.30
10.30
1
12.30
3.0
at 2.25 Cold Bath at 70°
1
3.15
5.45
1
4.45
10
9
8 a.m.
2 p.m.
1
3 "
4 "
at 3.30 Cold Bath I 75°
1
5 "
7 "
10
11 a.m.
11 p.m.
11
5 a.m.
5 p.m.
12
8 a.m.
8 p.m.
13
8 a.m.
11 p.m.

DESCRIPTION OF CHART IV.

Chart IV. illustrates the use of the cold bath in reducing the temperature of enteric fever. Three baths were given on the ninth, tenth, and the eleventh day of the disease, one on each day. No other anti-pyretic measures were adopted, and the patient made a speedy recovery. It will be observed that the temperature fell ten degrees after a bath at 70° Fahr. on June 8th, and that the thermometer did not again register 104° till twenty-four hours afterwards, when the third bath was given.

DESCRIPTION OF CHART V.

Chart V. illustrates the combined use of cold baths and quinine in the treatment of enteric fever. A bath on June 8th reduced the temperature from 104° to 99°; twenty-two hours later the temperature had again risen to $104\frac{2}{5}$, when a second bath reduced it to $98\frac{3}{5}$; it again rose rapidly, and in four hours had reached $104\frac{2}{5}$, when a third bath reduced it to $96\frac{2}{5}$; two hours later it had risen to 103, and four hours after the last bath to $103\frac{4}{5}$. The effect of the baths being so temporary, a dose of quinine (grs. xv.) was given, when a gradual fall took place during twenty-seven hours, the temperature then being $96\frac{3}{5}$. From this time the pyrexia was more moderate, ranging between 100° and 102°, only once reaching the 103° line.


June
104
103
102
101
100
99
98
97
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
Convalescent.
Bath.
Bath.
Bath

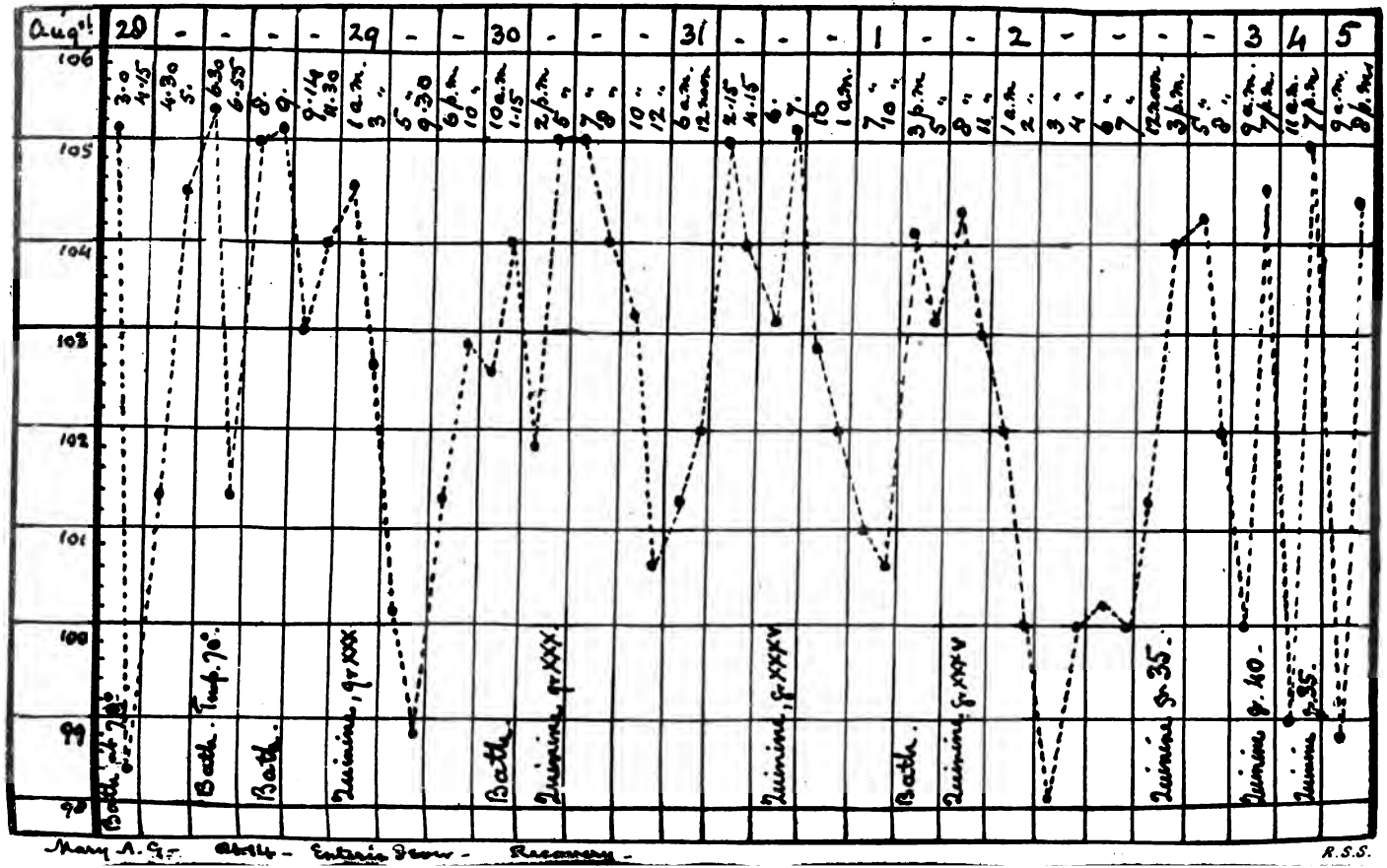

Aug.st
20
29
30
31
1
2
3
4
5
106
105
104
103
102
101
100
99
98
Bath at 7.30
Bath. Temp. 70°
Bath.
Quinine, gr. XXX
Quinine, gr. XXX
Bath.
Quinine, gr. XXXV
Bath.
Quinine, gr. XXXV
Quinine, gr. 35.
Quinine, gr. 40.
Quinine, gr. 35.
Mary A. G.
Bath - Entire Blow - Recovery -
R.S.S.

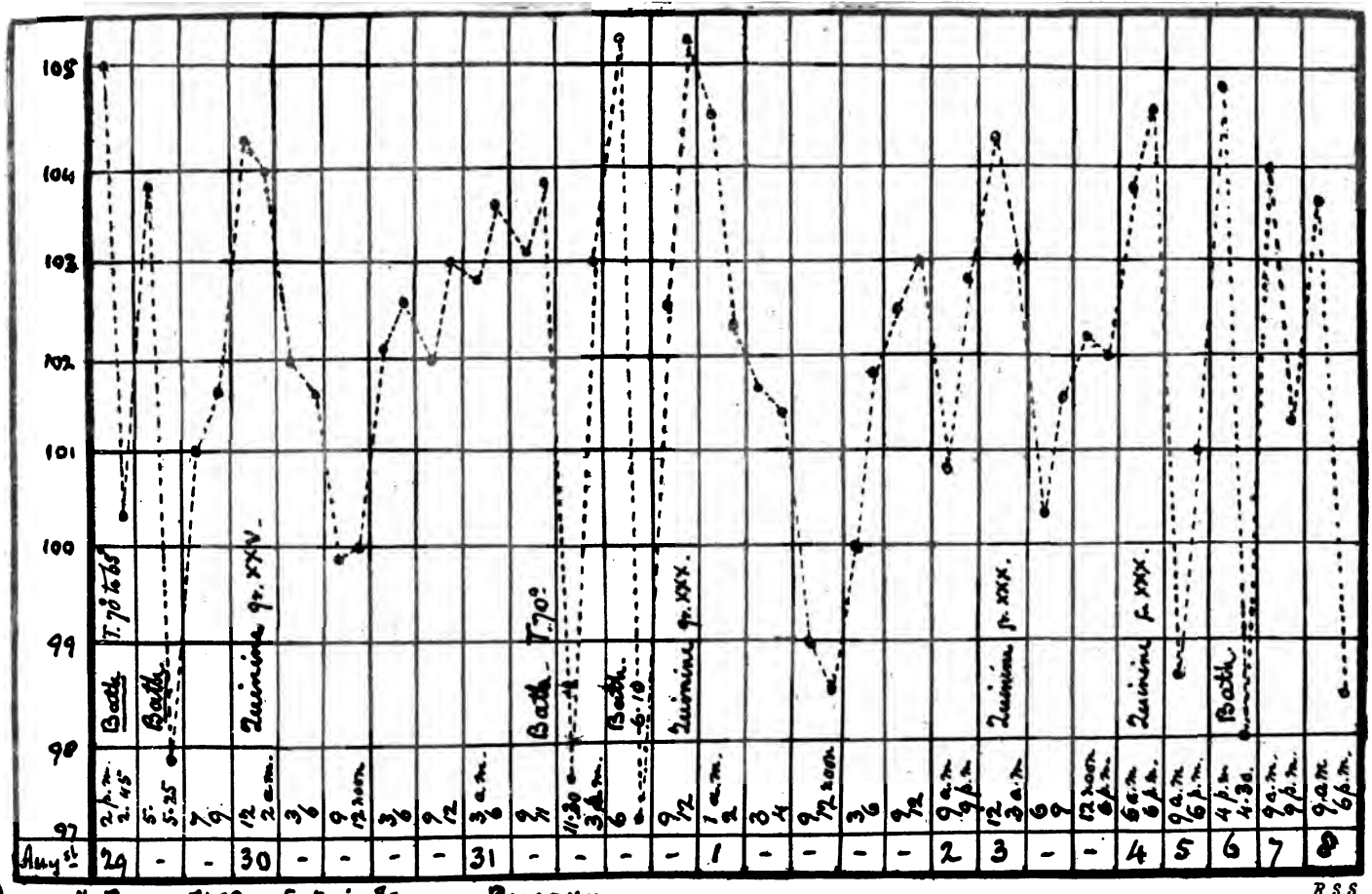
Chart VI

DESCRIPTION OF CHART VI.

Chart VI. illustrates the use of baths and quinine in a severe case of enteric fever. Five baths and seven doses of quinine were given within a period of eight days. It will be seen that the effect of the baths was very evanescent, and the temperature rose after the first two baths to a point above the 105° line; the third bath had little effect, accordingly quinine (grs. xxx.) was given, and the temperature fell from 104$\frac{3}{5}$ to 98$\frac{4}{5}$ in eight hours. Repeated doses of quinine reduced the pyrexia to an intermittent type, and the patient made a satisfactory recovery.

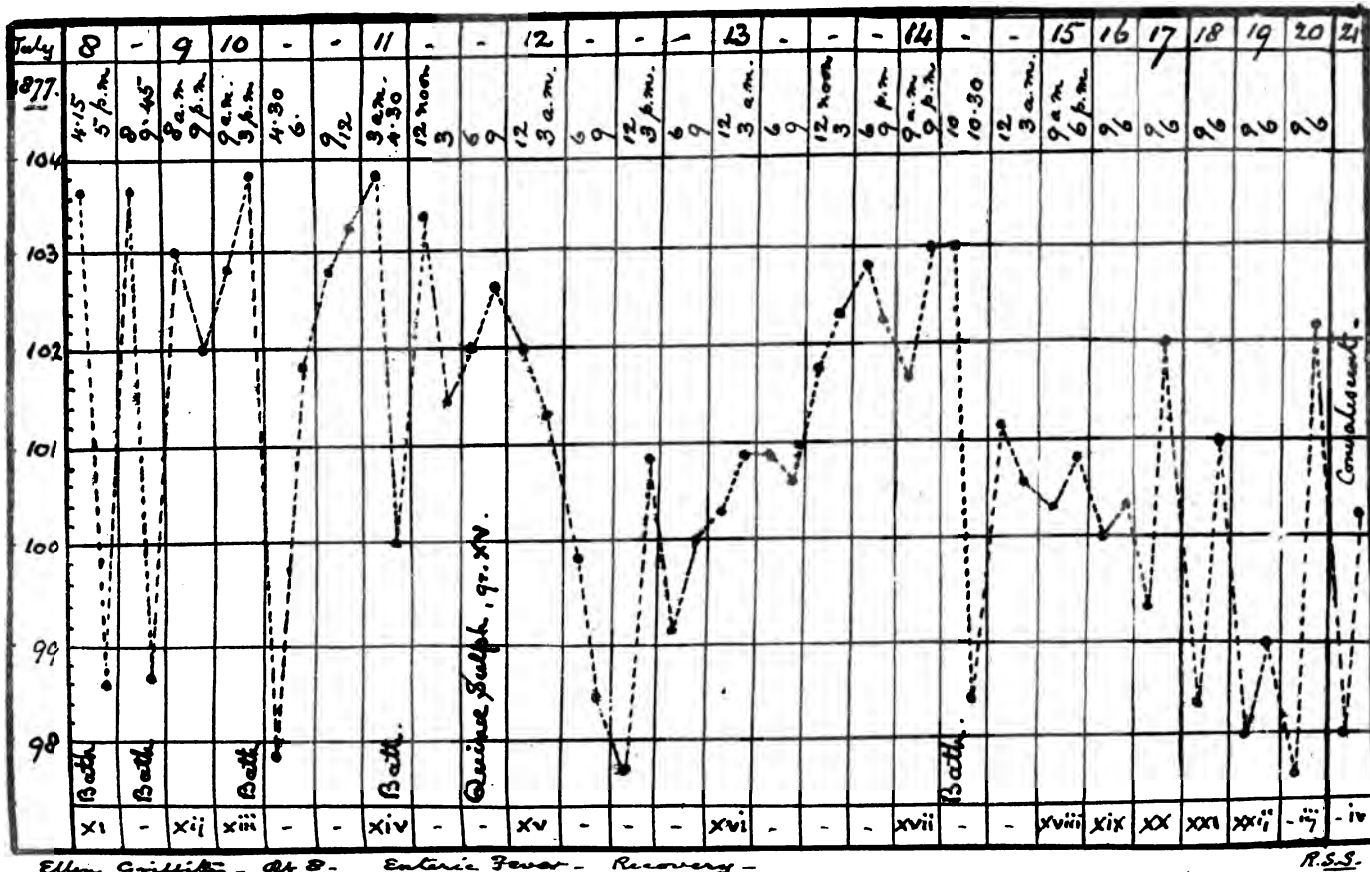
DESCRIPTION OF CHART VII.

Chart VII. also a case of enteric fever treated by baths and quinine. Five baths and four doses of quinine were given during a period of nine days; after this no further anti-pyretic treatment was necessary, and the patient made a good recovery in spite of a slight relapse. It will be seen that the temperature rose rapidly after the first two baths; nevertheless by means of them the necessity for quinine was postponed till evening. At midnight the temperature being 104⅔ a dose of quinine (twenty-five grains) was given, and the temperature fell to 99⅔ at nine o'clock following. No further treatment of pyrexia was adopted till the following day, when two baths (at 11.30 a.m. and 6.10 p.m.) allowed the second dose of quinine to be postponed till midnight. A larger dose (thirty grains) was given, and in twelve hours the temperature fell to 98⅔. Subsequent doses of thirty grains had afterwards a similar effect.



H.R. Ak13. Enteric Fever. Recovery.

R.S.S.



Ellen Griffiths - Ward 8 - Enteric Fever - Recovery -

R.S.S.

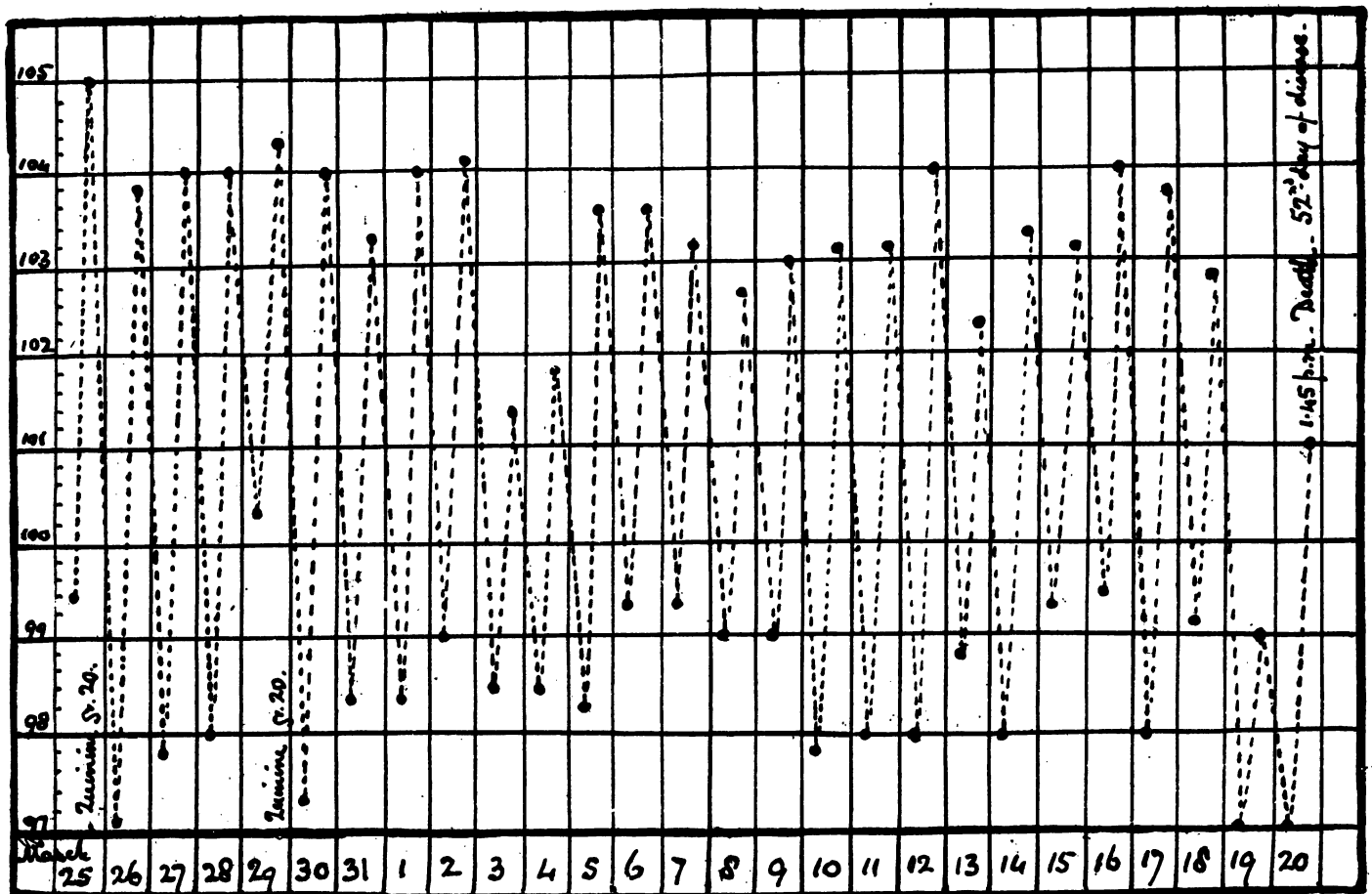
Chart VIII

DESCRIPTION OF CHART VIII.

Chart VIII. A case of enteric fever in a child eight years of age, in which the temperature was kept below the 103° line by means of five baths and one dose of quinine (grains xv.) Two baths were given on July 8th, one on the 10th, one on the 11th, and one on the 14th. The dose of quinine given on the evening of the 11th caused a fall from 102$\frac{3}{5}$ to 97$\frac{3}{5}$, after which a very very slow and gradual rise took place extending over three days, not reaching the 103° line till 9.0 p.m. on the 14th. After this, at 10.0 p.m., a bath reduced the temperature to 98$\frac{3}{5}$, and there was no subsequent rise to a sufficient height as to require treatment.

DESCRIPTION OF CHART IX.

Chart IX. represents the morning and evening temperature of a patient, aged 21, in whom pelvic cellulitis occurred; suppuration followed, and ultimately peritonitis from the discharge of pelvic abscess into the peritoneal cavity. For a period of thirty-seven days before death the evening temperature had been almost invariably above the 103° line, frequently up to the 104° line, and once it reached 105 degrees. The morning temperature was, however, usually below 99 degrees; the pyrexia was accordingly of an intermittent type. At the post mortem examination it was found that the voluntary muscles had not undergone the usual granular degeneration following prolonged and constant pyrexia: the heart fibre was only very slightly granular, and the kidney and liver cells were only slightly affected. On two occasions quinine in twenty grain doses was given, with the effect of exaggerating the usual nocturnal fall.



Maria S. Æt 21. Pelvic Cellulitis. Septicæmia. Death.

R.S.S.

Chart IX.

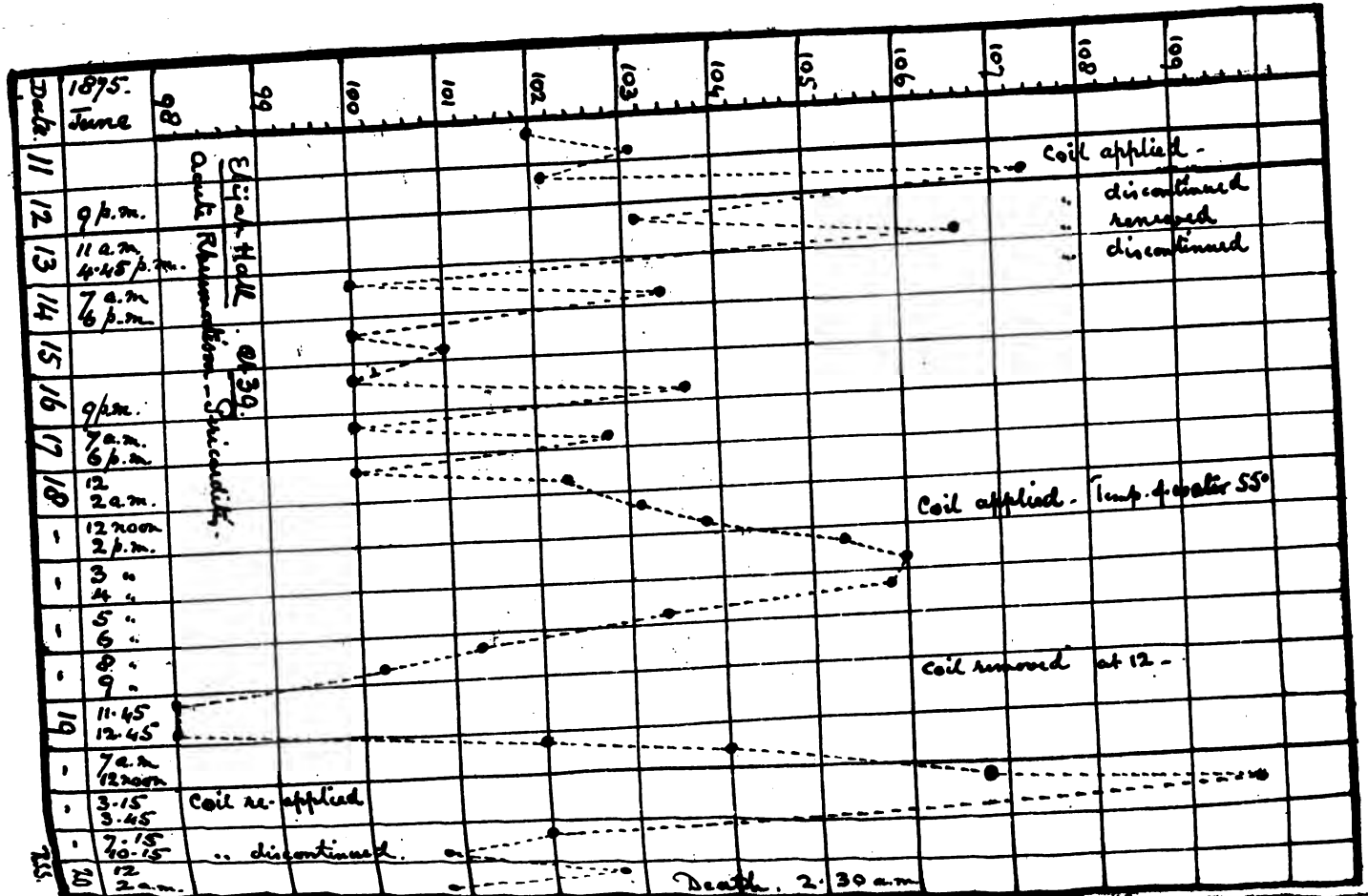

1875.
June
Date.
98
99
100
101
102
103
104
105
106
107
108
109
11
12
13
14
15
16
17
18
19
20
9 p.m.
11 a.m.
4.45 p.m.
7 a.m.
6 p.m.
9 p.m.
7 a.m.
6 p.m.
12
2 a.m.
12 noon
2 p.m.
3 "
4 "
5 "
6 "
8 "
9 "
11.45
12.45
7 a.m.
12 noon
3.15
3.45
7.15
10.15
12
2 a.m.
Elijah Hall. Æt 39.
Acute Rheumatism - Pericarditis.
Coil applied -
" discontinued
" removed
" discontinued
Coil applied - Temp. of water 55°
Coil removed at 12 -
Coil re-applied
" discontinued.
Death. 2.30 a.m.

Chart X.

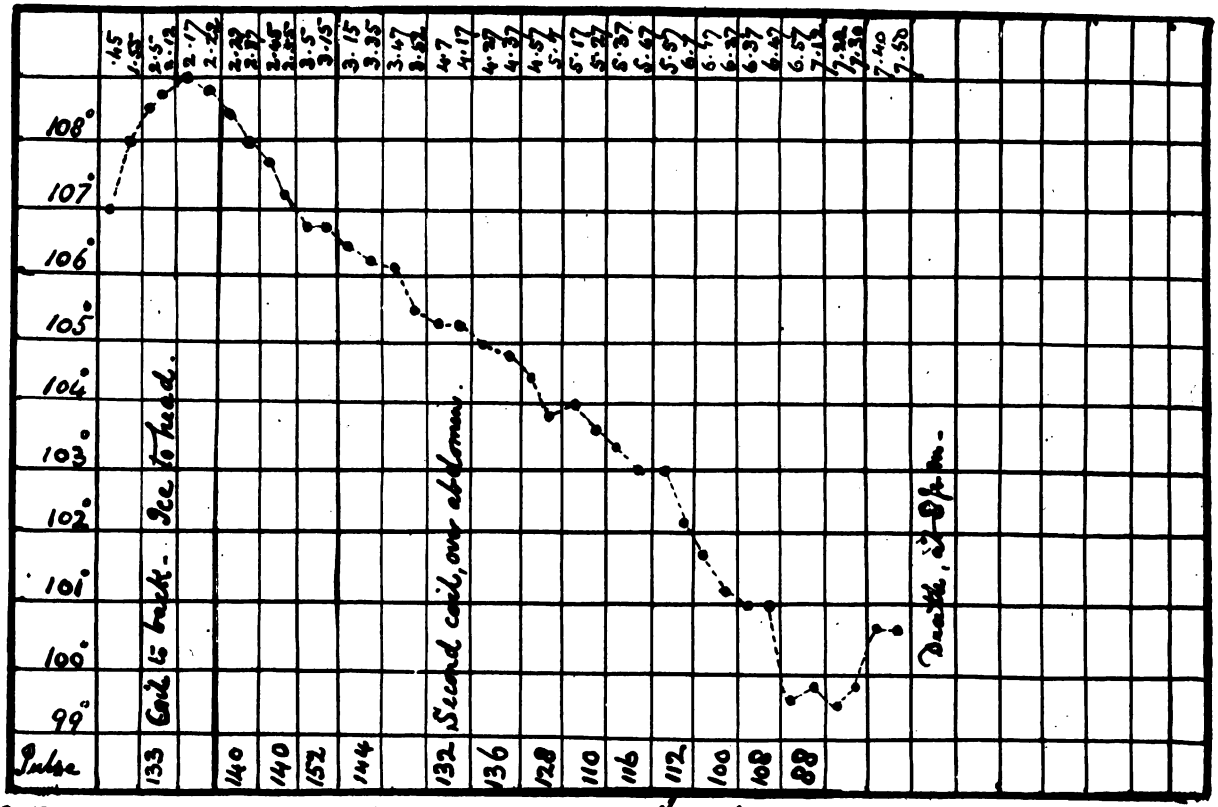
255.

DESCRIPTION OF CHART X.

Chart X. illustrates the effect of *Roberts's coil* on a case of acute rheumatism with hyper-pyrexia. The coil was first applied at 11 p.m. on June 12th, when the temperature was $107\frac{2}{5}$; it was removed at 9.0 a.m. on the 13th, when the thermometer registered $103\frac{1}{5}$. It was again applied at 4.40 p.m. on the 13th, the patient's temperature having risen to $106\frac{3}{5}$, and it was discontinued at 9.0 a.m. on the following day, the temperature having meanwhile fallen to 100 degrees. It was again applied at 5.0 a.m. on the 15th, and continued during the whole day, the temperature falling from $103\frac{2}{5}$ to 100 during its application. On the 18th the temperature rose again to 106° at 4.0 p.m., the coil had been applied an hour previously, and there was a gradual fall to 98° at midnight, when the coil was removed. Its removal was followed by a rapid rise, culminating in $109\frac{4}{5}$ at 3.45 p.m.; at 3.15 it was renewed, and at 8.45 the temperature had fallen to $100\frac{4}{5}$, when the coil was finally removed. Although the temperature had been reduced below 101° the patient died shortly afterwards.

DESCRIPTION OF CHART XI.

Chart XI. shews an unusually high temperature arising from sunstroke, and its reduction by means of iced water circulating through the india-rubber coil. The patient, a strongly-built robust looking man, had been working in the sun through the morning, and was brought to the Infirmary in a state of coma at about half-past one p.m. The skin was found to be exceedingly hot and dry. The thermometer registered 107°. The coil was immediately set in action, and ice was applied to the head and rubbed over the body. A temperature of 109° was registered at 2.17, but after this a gradual fall took place. A second coil was applied at four o'clock, and at 7.30 the temperature had fallen to 99⅔. There was no return of consciousness. The breathing continued to be stertorous, and the heart's action excessively weak. Death took place six hours after admission.



J. B. Æt. 45 - Heat-fever - July 15th 1876.

Chart XI.

R.S.S.

Period of first rise afterwards	26	hours.
Longest	54	,,
Shortest	15	,,
Time till next dose	50	,,
Longest	144	,,
Shortest	24	,,

Sickness occurred only twice in the sixteen cases. The average time of highest temperature was 6.30 p.m.; that of lowest temperature, 6 a.m. Salicylic acid had been found to act as an efficient anti-pyretic, but was more depressing in its after effects, and therefore less applicable than quinine, except in the pyrexia of rheumatism.

The charts appended illustrate the effects of various antipyretic agents under varying circumstances. (*See Charts I. to XI.*)

Dr. W. H. SPENCER proposed an adjournment of the debate, and at the following meeting on September 26th, 1877, re-opened the subject, as follows:—

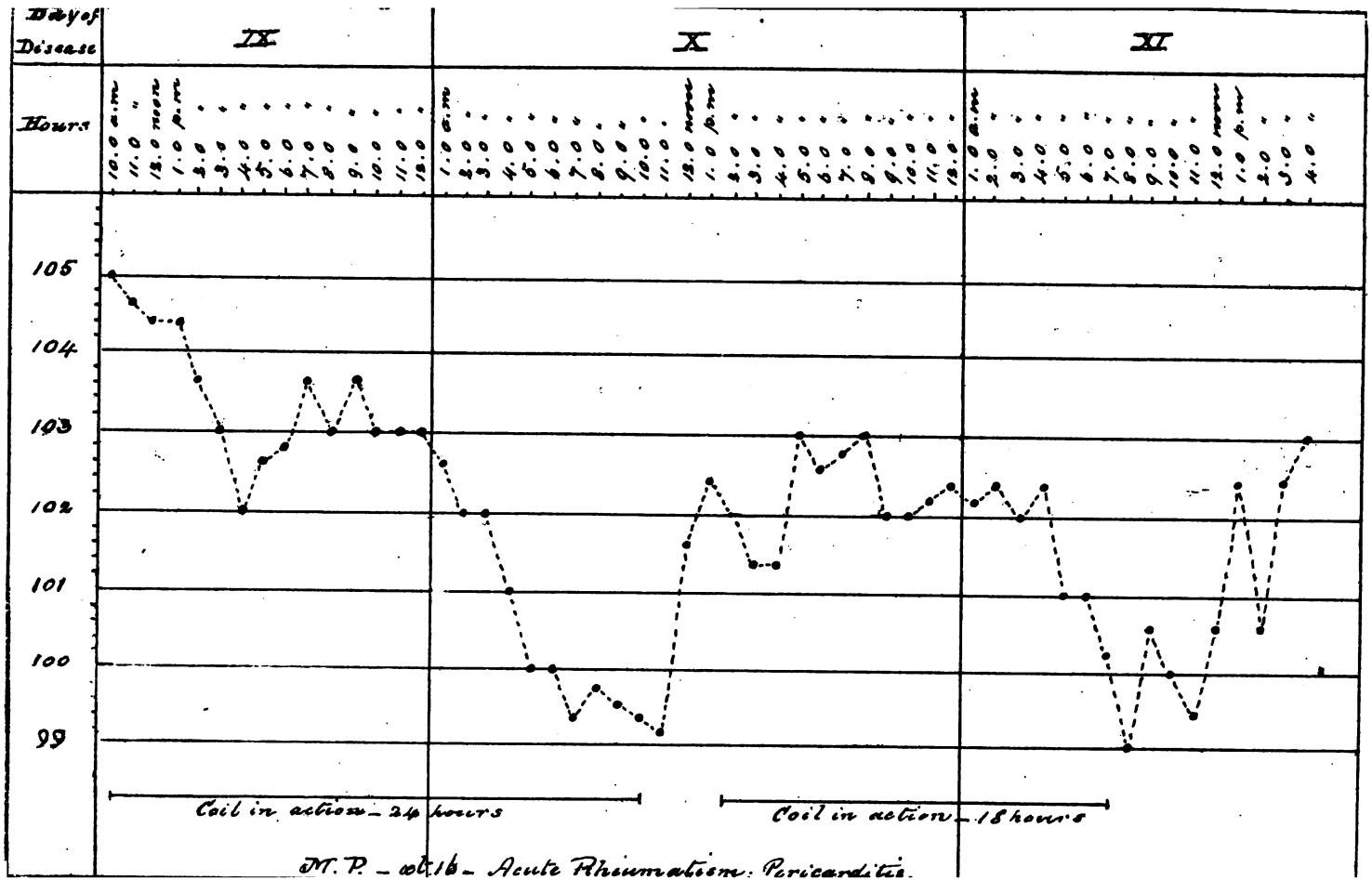
"A high body-temperature, however it may be brought about, must be set down as generally a serious and often a dangerous symptom. If it occurs and exists only for a short time, it is at least a retarding episode in the course of any disease; if it continues it is fraught with peril to the patient's life. There would seem, therefore, to be good reason why a high body temperature should be treated on its own account and in some way. The question is, when and by what means should high temperature be reduced?

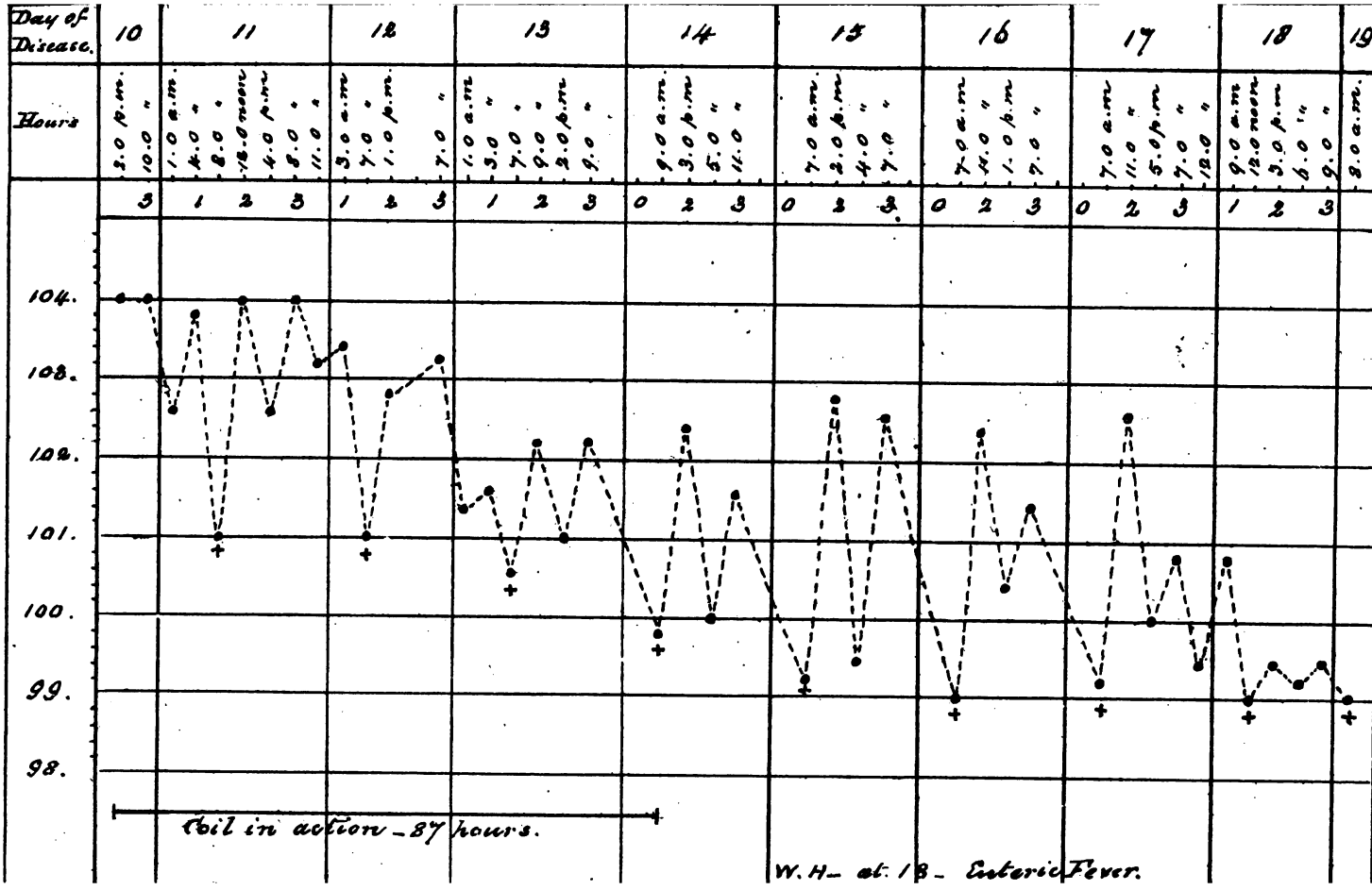
Doubtless the question involves a reference to the fever-process and to the disease, in the course of which the high temperature occurs. The nature of these would have much to do with the right choice of means. But our present knowledge in these

directions will give us little help. We have to abide by experience of different modes in different diseases and in different cases of the same disease. All we can say for a given disease or case is, that we know of nothing to make treatment of the temperature inadmissible in the particular disease and case; then we can reckon up the disadvantages of this or that mode of reducing temperature, and proceed to make observations as to the results of that mode we may select. Such observations, if exact, are a real contribution to our knowledge.

Practically, we are limited to two chief modes :—first, withdrawal of heat from the surface; secondly, control and regulation of heat-production. The second of these modes concerns the use of quinine and other febrifuges. This part of the matter I will leave to others, and will now confine my remarks to some observations I have made as to the use of means directed to the withdrawal of heat from the surface.

When heat is abstracted quickly from a large surface of the body, the line marking the active reduction of temperature is abrupt or even vertical, and its lower end lies as often as not in the region of collapse. Then follows a reactionary rise of temperature nearly, if not quite, to the height from which the temperature had been reduced. Such depression and subsequent reaction may be as serious or dangerous as a high temperature itself; and for my part, I hesitate on this account to expose my patients to the risks incurred by the use of such measures as the cold bath or wet sheet packing. It occurred to me some time ago to apply means for abstracting heat from the surface in such a way as to produce the smallest amount of depression and reaction. Accordingly I made use of Roberts's Coil and used in it water at about 60°, and continued the gradual withdrawal of heat by such means during long periods—registering the body temperature every two hours or oftener, day and night. The curves I got in this way confirmed the fact—which, however, hardly needed corroboration—that some re-action is the inevitable result of the use of any means for


Day of Disease
IX
X
XI
Hours
10.0 a.m.
11.0 "
12.0 noon
1.0 p.m.
2.0
3.0
4.0
5.0
6.0
7.0
8.0
9.0
10.0
11.0
12.0
1.0 a.m.
2.0
3.0
4.0
5.0
6.0
7.0
8.0
9.0
10.0
11.0
12.0 noon
1.0 p.m.
2.0
3.0
4.0
5.0
6.0
7.0
8.0
9.0
10.0
11.0
12.0
1.0 a.m.
2.0
3.0
4.0
5.0
6.0
7.0
8.0
9.0
10.0
11.0
12.0 noon
1.0 p.m.
2.0
3.0
4.0
105
104
103
102
101
100
99
Coil in action – 24 hours
Coil in action – 18 hours
M. P. – ward 16 – Acute Rheumatism. Pericarditis.


Day of Disease.
10
11
12
13
14
15
16
17
18
19
Hours
2.0 p.m.
10.0 "
1.0 a.m.
4.0 "
8.0 "
12.0 noon
4.0 p.m.
8.0 "
11.0 "
3.0 a.m.
7.0 "
1.0 p.m.
7.0 "
1.0 a.m.
3.0 "
7.0 "
9.0 "
2.0 p.m.
9.0 "
9.0 a.m.
3.0 p.m.
5.0 "
11.0 "
7.0 a.m.
2.0 p.m.
4.0 "
7.0 "
7.0 a.m.
11.0 "
1.0 p.m.
7.0 "
7.0 a.m.
11.0 "
5.0 p.m.
7.0 "
12.0 "
9.0 a.m.
12.0 noon
3.0 p.m.
6.0 "
9.0 "
2.0 a.m.
104.
103.
102.
101.
100.
99.
98.
Oil in action - 87 hours.
W. H. - at. 18 - Enteric Fever.

abstracting heat from the surface, so long as no measures are taken to control heat production. I think we may even formulate a law of cooling in these terms: the line of ascent after cooling is proportional in steepness and in length to the line of descent during cooling. We might describe the curve as V-shaped, and say that the two limbs will bend to approximate or diverge at the open ends according as the cooling is effected rapidly or gradually.

I will narrate briefly the observations made in two cases in which gradual cooling by means of Roberts's coil was practised.

A patient with acute rheumatism and pericarditis was kept on the coil for a period of twenty-four hours; in this period the temperature fell from 105° to 99·2°. After the coil was removed the temperature rose in three hours to 102·4°. Then the patient was placed on the coil again, and kept on it for a period of eighteen hours; in this period the temperature rose at first to 103°, and thence fell to 99°. After the coil was removed the temperature rose in five hours to 102·4°, and in eight hours to 103°. The amount of urea excreted was determined in this case throughout its course, and it was found that the percentage amount was half as much again during the cooling period as it had been during the exhibition of alkalies for some time prior to the cooling. (See Chart No. XII.)

In another case, a patient with enteric fever was kept in the coil for a period of eighty-seven hours. The results are shown in Chart No. XIII. Thecharacteristic feature of a typhoid curve is the diurnal exacerbation and remission of temperature. If the temperature is registered often enough, we shall find that in an average and uncomplicated case there are three exacerbations, and three corresponding remissions in the twenty-four hours. These are landmarks to us whereby we may estimate the course of the disease. Since gradual and moderate cooling does not obliterate these landmarks, as rapid or excessive cooling does, I have been able to preserve in the chart the general features of the entire curve, and so indicate the modifications produced in it by the mode of cooling adopted.

The general course of the disease in this case was fairly typical of an uncomplicated and not severe case. The coil was put in action on the tenth day of the disease, the temperature being then 102°, and there being present the eruption, some delirium, and moderate diarrhœa. It will be observed that on the eleventh and two succeeding days (temperature having been registered every two hours) the three exacerbations and remissions were well marked—numbered 1, 2, 3, in the third horizontal column from the top in the chart. The great morning remission (these remissions are marked + in the chart) on the fourteenth day was below 100°. The action of the coil was suspended at this time. From this time the course of the disease seems to have been favourably modified by the cooling; the first diurnal exacerbation disappears (its apparent re-appearance on the eighteenth day does not, I think, count for anything); the great morning remission reaches 99°, and maintains its low point; the third diurnal or evening exacerbation is lowered day by day, with one exception (the fifteenth day); the temperature regained a normal course after the eighteenth day—an early reversion to the normal even in a mild case. But there is one peculiarity in the course of the temperature after the cooling which is, I think, important and suggestive. During cooling the second or midday exacerbation had never reached a higher point than the third or evening exacerbation; after cooling it was higher than the evening rise, and reached on four successive days a point which must strike the most casual observer. This second diurnal rise seems altogether out of place, and is (especially when taken together with the third rise of the fifteenth day) suggestive of a re-action somewhat delayed and drawn out in correspondence with the drawn out period of cooling. When, in any case of high temperature, cooling is resorted to, I think it is better on the whole to cool gradually. I am not prepared, however, by any means to pin my faith to Roberts's coil; the apparatus is difficult to manage, and it is quite possible to cool gradually by means of a bath. Regard for the patient and such

experience as I have had and seen would indicate to me the use of an apparatus similar in principle to Robert's coil, but permitting easy adjustment to different parts of the surface, and an uninterrupted flow of water at any temperature we please."

Dr. E. MARKHAM SKERRITT said that cases where pyrexia was marked might be divided into two classes :—

i.—Where treatment was advisable.
ii.—Where it was imperative.

With reference to the first class he would only say that he entirely agreed with Dr. Smith's remarks on the importance of controlling the temperature, even when it was not so high as in itself to directly endanger life; and that he had found both salicin and salicylic acid act powerfully as anti-pyretics. It was said that relapses were frequent in rheumatic fever when treated by these drugs; but from his own experience he believed that relapse would be far less often met with if the use of the anti-pyretic was continued for some days after the marked effect had been produced. The advocates of the old treatment by salines would think their favourite remedies much maligned if they were discontinued as soon as they were apparently taking effect, and are then held responsible for the after course of the disease. And in the case of typhoid fever it was important to remember that there was a definite local lesion that must run a more or less protracted course, and that therefore it was not reasonable to expect the disease to be cut short, as rheumatic fever might be.

With regard to the second class, he would place in it those formidable cases, sometimes met with in rheumatic fever and other acute diseases, where there set in a rapid rise of temperature to a height incompatible with life, accompanied by profound nervous disturbance. Whatever the pathology of this condition of hyperpyrexia, it was well known that death invariably resulted if no treatment was adopted, and that the only treatment that had proved effectual was the use of the cold bath, as introduced by Dr. Wilson

Fox, inasmuch as other anti-pyretics—such as icebags, the water coil, quinine—had not time to act. (It was to be observed, however, that in some diseases a very high temperature might be of less immediate serious import—*e. g.*, in ague, relapsing fever, pyœmia, tubercular pneumonia.) The following case was a typical illustration of hyperpyrexia, and of its only remedy.

A man, aged 36, was admitted into the Bristol General Hospital with a well-marked first attack of rheumatic fever. On the evening of the 6th day, Mr. Harsant, the house-surgeon, found him delirious, with a temperature of 108°. He at once put him into a cold bath, but as the man was apparently dying from collapse took him out in fifteen minutes, when the axillary temperature was 104°. Ten minutes later it was 102°; within two hours it was 105°, and the patient was again violently delirious. At 10.30 p.m., Dr. Markham Skerritt found the patient delirious and struggling; the surface was very dusky and pungently hot, the pulse very small and rapid, only just perceptible at the wrist; the axillary temperature was 108°. (From this time the temperature was taken in the rectum). The patient was again put into the bath, and in ten minutes the temperature was 109·5°. The result was as follows:—

Time in bath—38 minutes.

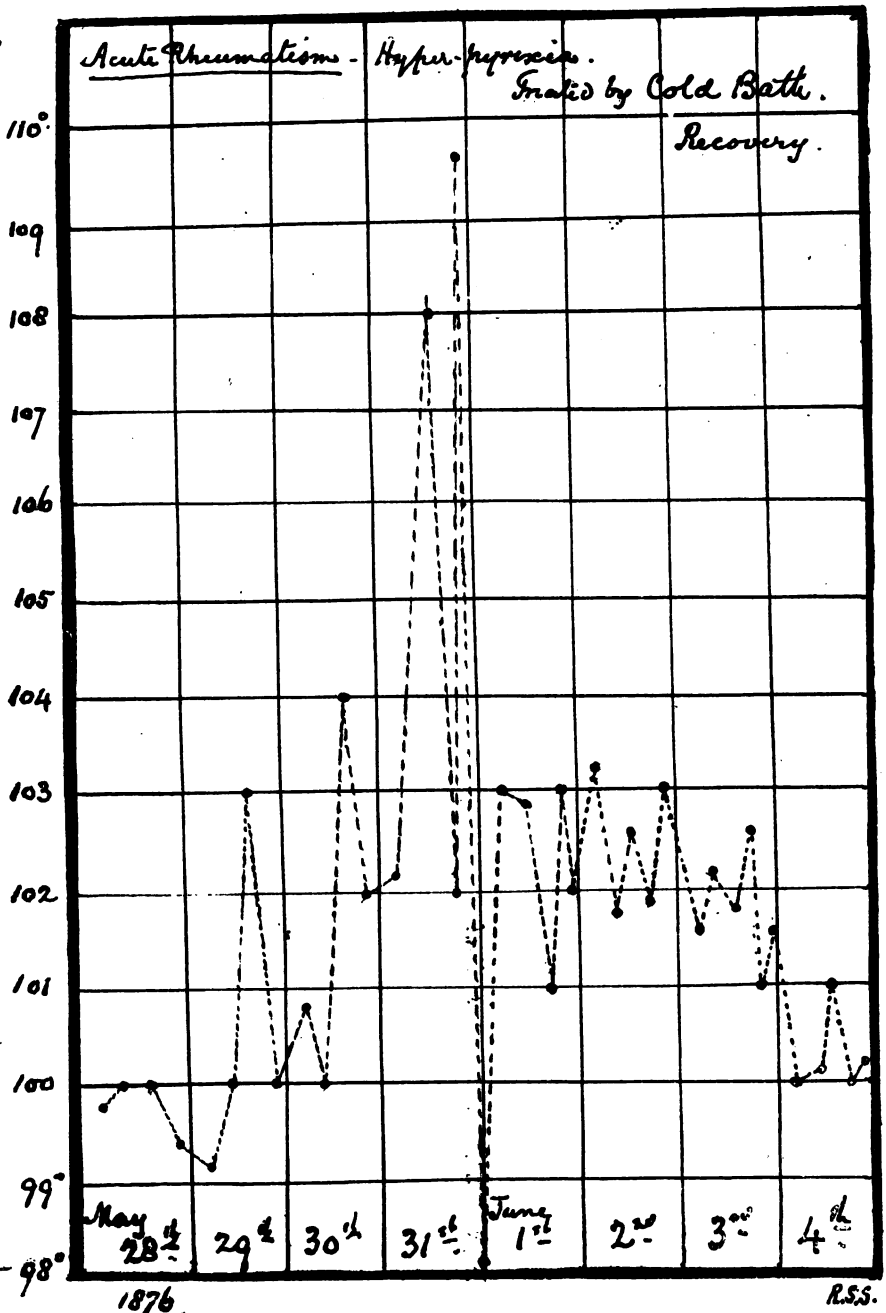
Fall of temperature in bath—from 109·5° to 102°; or 7·5° in 28 minutes.

Fall of temperature after removal from bath—4° in 10 minutes.

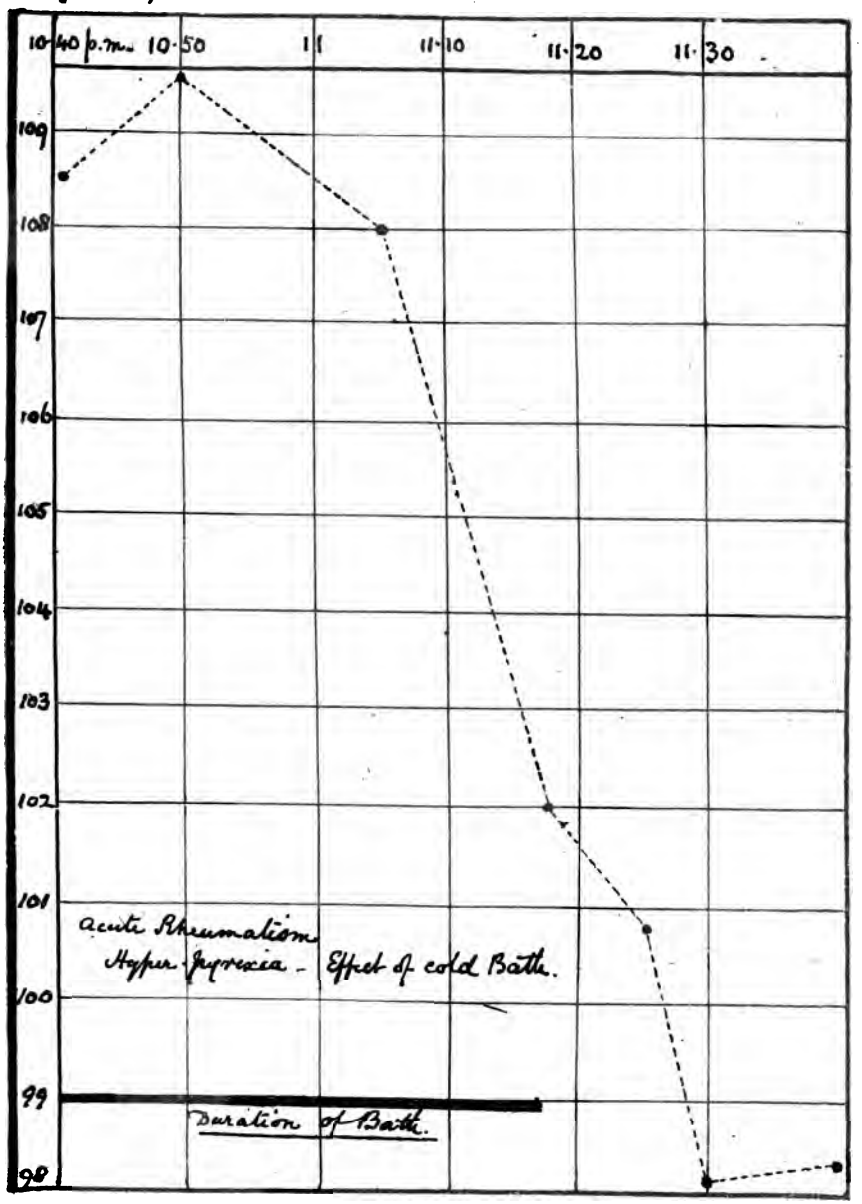
Total fall—from 109·5° to 98°; or 11·5° in 40 minutes.

Bath cooled from 88° to 61°.

While in the bath the patient looked as if *in articulo mortis;* he was pulseless at the wrist—the heart was beating 156—most of the time struggling; every now and then he became quiet and almost comatose—in fact, so certain did death appear, that only the conviction that it was his only chance made it possible to persevere with the bath. Twenty minutes after removal to bed he was


Acute Rheumatism - Hyper-pyrexia.
Treated by Cold Bath.
Recovery.
110°
109
108
107
106
105
104
103
102
101
100
99°
98°
May 28th
29th
30th
31st
June 1st
2nd
3rd
4th
1876.
R.S.S.

May 31st 1876



R.S.S.

perfectly quiet, comfortable, and almost rational, and the pulse was 108. From this time there was steady though slow progress towards convalescence. In similar cases quinine had generally been given in large doses for some time after the bath; but in the belief that this drug is not an essential adjunct, it was not ordered; in fact, no medicine at all was given for nearly three weeks, and although the temperature several times rose to 103°, no further anti-pyretic treatment was adopted. This case was of interest as showing the remarkable power of the cold bath in very rapidly reducing a hyperpyrexial temperature, and also as proving that there might be no need for any further special treatment. (The two accompanying charts show respectively the course of the temperature throughout the disease, and the precise effect of the cold bath).

Dr. W. J. Fyffe said—"As regards the treatment of fevers by quinine, I may observe that this is no new remedy. Thirty years ago, when I practised in the West Indies, quinine in doses of twenty grains was in full use in the treatment of fevers.

In that climate the fevers are all of short duration, very rapid and intense in character, and very fatal. Yellow fever proves fatal in a few days; the acute congestive malarial fever of Jamaica, though not so fatal as yellow fever, seldom lasts more than a week. In all these fevers quinine, as far back as 1849, was the sheet anchor. It is true we did not recognise it as a special anti-pyretic; we believed rather in its specific powers as an antidote against malarial poison.

Dr. Davy, Inspector-General of Hospitals, the brother of Sir Humphrey Davy initiated the treatment by quinine and calomel of yellow fever during the epidemic of 1848.

The calomel was found afterwards to be useless, but quinine in large doses frequently repeated is the treatment adopted up to the present day in acute tropical fevers.

Again in sunstroke, which some regard as fever of congestive character, and where the most intense hyperpyrexia exists, quinine

has been frequently and usefully employed, and latterly in several cases hypodermically, with remarkably good effect. This mode of using quinine is however not unattended with danger; several fatal cases of tetanus having occurred in India after this mode of administration,—consequently the hypodermic method had been rather discountenanced.

There is one plan of treating high temperature, which I think has not been mentioned, namely—the repeated injection of iced or very cold water into the rectum and large intestines. This procedure is often adopted in cases of sunstroke. It was strongly advocated by the late Dr. Parkes, and has been revived by Dr. Rutenburg, of Vienna, who considers that it is quite as efficacious in reducing high temperature as the cold bath. There are, of course, cases where this treatment would be inadmissible—for example, where ulceration of the lower bowel is present; but when we reflect upon the large surface of blood flowing in the iliac and mesenteric veins and vena cava, which may be cooled by close proximity to the intestine thus filled with cold fluid, the procedure appears worthy of consideration."

Dr. Henry Waldo spoke of the difficulty in giving baths, more especially to children, and expressed his preference for cold sponging. He gave particulars of two severe cases of enteric fever in which the temperature had been reduced by means of cold baths and quinine, both of which cases recovered.

The debate was re-opened on November 28th, 1877, by Dr. E. L. Fox. He alluded to the implication of the nervous system in cases of pyrexia, and considered that implication of a heat-centre is shown by the condition of paresis of the heat-regulating function, and this paresis is intensified by the circulation of over-heated blood. Dr. Fox would consider that a temperature of 105° or over in cases of pyæmia, sunstroke, erysipelas, &c., is one which requires treatment, but the cold bath and its modifications may be supplanted to some extent by other agencies; he had found the

Warburgh's tincture to have an unquestionable effect. He believed that Dr. Roberts's coil is preferable to the cold bath, and that the injection of water into the rectum had been found to act efficiently.

With regard to the other class of cases in which treatment of the pyrexia may be considered advisable but not imperative, Dr. Fox would not interfere with acute rheumatism or typhoid if the temperature did not amount to 105°, for the following reasons:—that such treatment would probably prolong the course of the disease; that it would increase the tendency to relapse; that the systematic treatment of such pyrexia is surrounded by difficulties in private practice, more especially among the poor; that it necessitates the administration of larger quantities of stimulants; and that it can be shown by statistics to be unnecessary.

Dr. J. Greig Smith gave an abstract of the results of cold baths and quinine in thirty-two cases, in which each had been employed. These were cases of enteric fever. The salicylates, except in rheumatism, were not found to be of service. He preferred the bath to Roberts's coil, as he believed it to be more efficient and less troublesome. He found that most patients became more and more tolerant of the bath, and more particularly so of the quinine; the dose of the latter needed to be increased to keep up a given effect. In cases of feeble heart the quinine would alone would be advisable. Delirium is influenced for the better by both remedies. Cinchonism was found to be a very evanescent and insignificant factor. Sickness had occured only six times.

The following is a summary of the thirty-two cases of each method.—

BATHS.

Average temperature for which bath was given -	104°.7
Fall in degrees - - - -	6·05
Time in falling - - - -	30 minutes
Height of rise after - - -	103·36
Time till next rise - - -	6·1 hours
Temperature of bath - - -	69·6
Time in bath - - - -	14 minutes

QUININE.

Average temperature for which quinine was given-	104°7
Fall in degrees - - - -	5·74
Time in falling - - - -	15·98 hours
Height of rise after - - -	103·18
Time till next rise- - - -	23 hours

Dose, about 35 grains.

Dr. SHINGLETON SMITH, in reply to the criticisms and comments made, pointed out the fact that no one had questioned the necessity for the treatment of pyrexia; all agreed that cases of hyper-pyrexia needed immediate and energetic anti-pyretic measures, but in cases of more moderate temperature, opinions would give a varying limit as to the line where treatment becomes a necessity. He expressed his belief that in typhoid and other moderate pyrexias, the difficulties in counteracting the tendency to undue elevation of temperature would be surmounted, and felt convinced that the cold bath by itself, or alternating with quinine, would be found to act most efficiently, and to give the most satisfactory results.

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